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THE EOSINOPHIL RESPONSE TO EXERCISE STRESS IN YOUNG MALE SWIMMERS

BY

MICHAEL VICKOVIC

(C)

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDY AND RESEARCH
IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE
OF MASTER OF SCIENCE

IN

PHYSICAL EDUCATION

EDMONTON, ALBERTA

FALL, 1979

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "The Eosinophil Response to Exercise Stress in young Male Swimmers" submitted by Michael Vickovic in partial fulfillment of the requirements for the degree of Master of Science.

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Supervisor

.....

.....

Date ..June 12 1979.....

DEDICATION

I wish to dedicate this piece of research to all those who gave unselfishly of their time and effort in helping me "get it together"; it would have been impossible to complete without your encouragement and support.

ABSTRACT

The purpose of this investigation was to determine if there is a chronic eosinophilic response to training, that is, exercise stress. If so, does the intensity, duration and/or total work output affect this response. Differences between pre and post-exercise eosinophil values were also examined and possible causes for those differences explained.

Twenty nine male swimmers volunteered as subjects for this study. Pre-training (basal) values were obtained for the eosinophil counts (EOS), the total white blood cell counts (TWBC), maximal oxygen consumption (MVO_2), percent body fat (%BF) and maximal swim times (MS) for 100, 200 and 400 metres. The latter three were used as criterion tests. After the two weeks of blood sampling during the no-trining period (to determine basal values for Eos and TWBC) the subjects were assigned to four experimental groups to complete six weeks of training. Samples were obtained every Monday, Wednesday and Friday of each week, both pre and post exercise.

Anova techniques were used to determine differences between groups on the above variables and the multiple rangetest by Scheffe was used to locate significant mean differences.

As a result of this investigation the following may be concluded: There is both an acute and a chronic EOS response to the stress of swimming training over a period of six weeks; this response is indicated by varying degrees of eosinopenia; adaptation to the stress of training seems to occur at approximately the fifth week of gradually increasing total work output; the TWBC response to exercise

stress over a period of six weeks is far less evident than the EOS response; and finally, with basal values for comparison, the EOS response to the stress of exercise reflects fairly accuratly the level of adaptation of the body even over just six weeks of training.

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Thanks, also to Dr. Jim Hill and Dr. Murray Smith whose encouragement helped during periods of doubt and whose enthusiasm became infectious.

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CHAPTER I

INTRODUCTION

Interest in the eosinophil as an indicator of stress dates back to 1846 (Vaughn, 1953). It's behavior in the vascular pool is looked upon as an index of adrenal cortical function (Weber, 1971) as well as a valuable asset in quantifying degrees of stress resulting from physical exercise (De Lanne, et al, 1960). Wake (1953) used the eosinophil response to exercise to determine the acute reaction of the adrenal cortex and was able to conclude that certain levels of exercise cause activation of the adrenal cortex in man. Weber (1971) has postulated that the circulating eosinophil count is the most accurate index of changes in output of adrenal steroids and since the adrenal cortex makes it possible for various regulatory systems to expend the additional effort necessary for homeostatic adjustments, it would seem appropriate to study the behavior of the eosinophilic leucocyte to predict and understand maximal performance (Weber, 1971).

Muscular activity produces significant changes in white corpuscles. The reactions of white blood cells (WBC) depend upon the duration and intensity of the exercise, the individuals' fitness level and the recovery time prior to sampling (De Lanne, 1960). The production of new cells, the accelerated destruction of circulating cells redistribution of leucocytes among various organs and modification in the plasma concentration have all been suggested as

mechanisms which produce this myogenic leucocytosis (De Lanne, 1960).

Of the studies reviewed by this author which used exercise as the stressor, all examined the acute response of either total WBC counts and/or eosinophil counts to either submaximal or maximal exercise stress. None of the related literature reviewed deals with the chronic response of the eosinophil to a period of training in man. It may be that the chronic response of the eosinophil is a more reliable indication of the body's ability to adapt to the stress of training. (Refer to Appendix A regarding the statement of hypothesis.)

Keeney (1960) found that rats trained for six weeks showed an increase in the magnitude of the eosinophil response to exercise, that is, a greater eosinopenia was exhibited. He suggests that a period of physical training increases the functional capacity of the adrenal cortex which is concomitant with the well-known enlargement of the adrenal gland due to training. Frenkl and Csalay (1962) studied the effects of forced swimming on rats over a similar period of time and largely agreed with the results of Keeney regarding increased size/weight of the adrenal gland. But, despite continued increases in weight during the sixth week, the hormone level showed a trend towards control base-line values. This, they concluded, confirmed the morphological and functional dissociation of the adrenal cortex in rats under chronic stress. With a drop in hormone production there should be a drop in eosinophil levels, also, if the latter are indeed a good index of adrenocortical function. But what does the decrease in hormone level indicate, exhaustion or adaptation?

Frenkl and Csalay (1966) suggest that normalization of the adrenal hyperfunction so characteristic of the first phase of training would represent a beginning of adaptation.

Looking at another aspect of the problem, Gofton et al (1953) points out that in using the chamber count method of counting eosinophil cells, the precision with which eosinophil levels may be determined is not great, yet changes in these levels, under fairly well standardized conditions, provide an index of adreno-cortical activity which is sensitive and has some reliability. Whereas the differential count performed on a smear has less reliability and accuracy due to the limited number of cells sampled.

Fortunately, the precision with which eosinophils may be counted has improved with the advent recently of the Hemolog D, a hematological analyzer which samples ten thousand cells and differentiates between the various types of white blood cells. With the aid of this instrument more precise evaluations of eosinophil changes are possible.

The use of eosinophil response to the stress of training has been examined by various researchers (Chin et al., 1971; Gofton et al., 1953; Keeney, C.E. 1960; Persky, H., 1953; Schneider, E.C. et al., 1915; Sealander, J.A. et al., 1970; Steadman, R.T. et al., 1969; Suzuki, T., et al., 1967; Wake, R.F. et al., 1953; Weber, H., 1971; Weswig, P.H. et al., 1974). Despite differences in the interpretation of results there seemed to be a general consensus as to the direction and time lapse of the eosinophil response to exercise stress. Unfortunately, more specific information

is required to enable researchers to more accurately assess the value of the eosinophil count and its relationship to both the total white blood cell count and the degree of training stress. Is there a chronic eosinophilic response to training? Does adaptation to training produce a normalization of the eosinophil response? Which factors contribute to the eosinophil response; intensity, duration or total work output? Is there a significant difference between pre- and post-exercise eosinophil values?

With these questions answered one could then determine the effectiveness, and possibly the accuracy of using the eosinophil count as an indicator of adaptation to exercise stress. If a positive answer to this question is found then the eosinophil response to the training load would be a valuable aid in the preparation of athletes, especially swimmers for major competition where a successful "taper" preceeded by maximal preparation is of the utmost importance in achieving maximal performance on a specific day.

Needless to say the difference of opinion and interpretation of research findings concerning the reaction of the adrenal cortex to muscular exertion as discussed in Chapter II, has implications for the possible use of the eosinophil count as an index of exercise stress due to the close relationship between eosinophil level and adreno-cortical function. It is hoped that this study will contribute some positive findings to this controversy.

A. STATEMENT OF THE PROBLEM

The purpose of the study is to determine:

1. Whether or not there is a chronic eosinophil response to training.
2. If so, does the intensity, duration, and/or total work output affect this response.
3. If there is a significant difference between pre- and post-exercise eosinophil levels.
4. If so, is this difference attributable to intensity, duration and/or total work output.

B. DEFINITION OF TERMS

Eosinophilia:	an increase in the number of circulating eosinophilic leucocytes.
Eosinopenia:	a decrease in the number of circulating eosinophilic leucocytes.
Exercise Stress:	muscular activity which causes regulatory adjustments in order to maintain homeostasis.
Duration:	time required to complete a task/exercise.
Intensity:	a combination of speed and resistance.

Relative Intensity: that intensity peculiar to an individual reflected in his/her heart rate response to a specific work load.

Taper: a period of reduced training prior to a major competition designed to allow the athlete to recoup his resources for a maximal effort.

CHAPTER II

A REVIEW OF LITERATURE

A. EARLY RESEARCH

The eosinophil was probably first described by Wharton Jones in 1846 when he included a description of "coarse granular cells" in his report to the Royal Society (Cline, 1975). In 1878, Ehrlich was able to stain these granules red with eosin, an acid dye, hence the name eosinophil. Thus the study of the role of the eosinophilic leucocyte began. The cell was so attractive to morphologists that in 1914 Schwartz was able to write a monograph about it that contained 2,758 references (Honsinger et al., 1972). The close relationship between the adrenal gland and eosinophils was observed by Dalton and Selye in 1939. Thorn (1948) and co-workers developed a test for adrenal cortical insufficiency using the eosinophil as an indicator.

Despite the well-documented association of this cell with allergic diseases, its specific functions in the body's economy remain largely unknown (Cline, 1975). Recent interest in eosinophils has centred around their association with parasitic infections, histamine, the antigen-antibody complex, the mast cell as well as allergy. As a result, the major roles postulated for the eosinophil are sequestering immune complexes and limiting inflammatory reactions

(Cline, 1975). In addition to allergens and foreign proteins, protein breakdown products such as polysaccharides, can cause eosinophilia (Olson, 1968).

B. ORIGIN AND FATE OF THE EOSINOPHIL

The source of the eosinophil leucocyte is the bone marrow. Metamorphosis of an eosinophil is: undifferentiated stem cell.... myelo blast....promyelocyte....eosinophilic myelocyte....eosinophil (Miller, 1966). According to Osgood (1954), the life span of human eosinophils in the blood is eight to twelve days. Formation of an eosinophil in the bone marrow takes twenty four hours and remains in the marrow for three to four days. One-half of the cells leave the blood in the first hour to locate mainly in the lung, gastrointestinal tract and the skin (Donohugh, 1966).

The bone marrow responds readily and is able to begin replenishment of the marrow reserve within two days after eosinophilotactic stimulus. The formation of eosinophils has not been shown to occur outside of the bone marrow in normal man, but has been shown to occur in the spleen, thymus and lymph nodes of some laboratory animals (Honsinger, 1972).

The fate of the eosinophil and its toxic engulfed material is not known, according to Honsinger, who suggests that since eosinophils are found in most body secretions that this may be the only means of disposal. Destruction in the reticuloendothelial system has also been proposed (Honsinger, 1972) since inactivation of the

system by the administration of trypan blue leads to eosinophilia. After a short period of circulation the eosinophil ends up in the mucosal cells of the bronchus or the intestinal wall where (at least in the lung) it is eliminated from the body or is picked up in the lymphatics and returned to the blood which carries it to the spleen to be destroyed (Vaughn, 1953).

Rytomma (1962) demonstrated in studies on the rat that the greatest accumulation of eosinophils were found in the bone marrow, which exceeded the blood content by 300:1. Some eosinophils are found lining the lungs, the gastrointenstinal tract and the skin. These areas are repeatedly assulted by foreign material from outside and therefore show natural eosinophilia. Acland (1956) has shown a diurnal eosinophil rhythm to be present in man with the highest peripheral count being obtained around 6.00 a.m. There is then a normal reduction in this count of about 20% by 10.00 a.m. This decrease of eosinophils during the day and increase during the night is probably associated with the action of the pituatory-adrenal system, but the mechanism remains obscure. It does, however, point up the need to take into consideration the natural diurnal eosinophil rhythm when making eosinophil counts.

C. FUNCTION OF THE EOSINOPHIL

The functions of the eosinophil are not fully understood but its phagocytic activity is well known. Eosinophils have been shown to phagocytize microorganisms, red blood cells, and soluble antigens.

According to Litt (1964) and Sabesin (1963), it is not until after the antigen-antibody complex is formed that the eosinophil phagocytizes the complex.

Lysis of the eosinophil granules obtained from isolated eosinophils was found by Bosworth (1962) to be associated with the release of protein and perioxidase from the granules. The material released from the granules attaches itself to foreign matter, and thus enhances ingestion by mononuclear phagocytes.

Archer (1963) proposed that eosinophils antagonized the action of histamine and serotonin basing his findings on the eosinophilotactic property of histamine in the skin of ponies. Litt (1962) was unable to confirm these findings with the use of other species.

The phagocytic properties and enzymatic contents of the eosinophil cell allude to the role it plays in inflammatory response, like other leucocytes. But, also Hosinger (1972) proposes that the eosinophil serves a role in fibrinolysis. This is based on his findings that the number of eosinophils in the inflammatory exudate was dependent upon the concentration of fribrinogen or fibrin and on the fact that profibrinolysin activation can be considered the final common pathway for the action of many known eosinophilotactic agents such as antigen-antibody complexes and the inflammatory reaction associated with pepsin, ptyalin and trypsin.

The eosinophil may also play a role in modifying delayed hypersensitivity as observed in the retest reaction which occurs before circulating antibody is detectable and is inhibited by anti-lymphocyte serum (Honsinger, 1972).

D. EOSINOPHILIA

Litt (1966) found that eosinophilia is not a specific indicator for allergy alone, instead, it is a response to a variety of agents as well as antigen-antibody complexes. Eosinophilia denotes an increase in the number of eosinophilic leucocytes in the blood. The upper limit is about 4% of the total white cells which is normally about 7,000 per cubic millimeter giving a normal upper limit of approximately 280 per mm^3 (Douthwaite, 1967). Beeson and Bass (1977) tend to push this figure a little higher to about 350 per mm^3. Conditions most commonly associated with eosinophilia are allergy, parasitic infestations and skin diseases.

Some of the conditions of eosinophilia are summarized in Table 1. Hudson (1963) has shown that foreign protein injected into guinea pigs causes a significant increase in circulating eosinophils but not until after the fourth injection at five-day intervals. Barr et al (1960) have shown that heparin, also increases eosinophil counts perhaps due to the blocking of some eosinolytic substance by the heparin.

Eosinophilia is considered to be closely related to the release of histamine, the presence of foreign protein, and protein catabolites. Archer (1960) produced eosinophilia in test animals (ponies) by injecting histamine. The use of repeated injections of histamine to stimulate increased production of eosinophils was made by Zaidi (1956) who found that over a four day period there was a gradual diminution of circulating eosinophils following the

Table 1 Conditions Associated With Eosinophilia

- Drug reactions
  - Iodine sensitivity
  - Penicillin sensitivity
- Parasitotis
  - Trichinosis
  - Visceral larva migrans
  - Tropical eosinophilia
- Infections
  - Leprosy
  - Brucellosis
  - Tuberculosis
  - Fungal infections
  - Scarlet fever
- Malignancies
  - Carcinoma of the lung
  - Carcinoma of the ovaries
  - Carcinoma of the stomach
  - Hodgkin's disease
- Collagen disease
  - Periarteritis nodosa
  - Rheumatoid arthritis
- "Cutaneous"
  - Pemphigus
  - Dermetitis herpetiformis
- Loeffler's syndrome
- Farmer's lung
- Asthma, hay fever
- Chronic eosinophilic pneumonia
- Leukemia
  - Chronic myelocytic
  - Eosinophilic
- Eosinophilic endocarditis (Loeffler's endocarditis)
- Unknown causes
  - Idiopathic
  - Sarcoidosis
  - Familial

(p. 114, Cline, 1975).

initial increase, in spite of increased dosages of histamine. It is conjectured by Olson (1968) that histaminase may be formed as a delayed response, decreasing the need for eosinophils. Archer (1963) reasons that following local histamine release from stressed tissue, chemotaxis occurs resulting in local eosinophilia. This response is immediate and localized and should not be confused with the generalized peripheral eosinopenia (drop in eosinophil counts) found following stress and surgical trauma (Olson, 1968).

Eosinophilia may occur in the absence of any apparent illness or disease and is sometimes hereditary (Honsinger, 1972), rarely it may be extreme and functionally malignant without any signs of abnormal immunological stimulation.

E. EOSINOPENIA

The endocrine system forms a link in the regulation of blood formation and destruction. Adrenal-cortical steroids, ACTH and stress usually cause eosinopenia as well as lymphopenia and neutrophilia. In the Thorn (1948) adrenal function test the number of peripheral eosinophils reflects the function of the gland. Eosinophils disappear when the subject is given ACTH or cortisone in the presence of a functional adrenal cortex. Whereas in the case of a damaged adrenal cortex, ACTH will not cause eosinopenia (Cornelius and Kaneko, 1963). Hoffer (1955) points out that in the use of the Thorn test the total diurnal eosinophil count must be established in order to determine a base line for determining

adrenocortical activity.

Hunter et al (1955) noted that adrenalin causes maximal eosinopenia in two to four hours. In man, adrenalin does not cause an increased adrenocortical secretion. Aschkenasy (1959) states that blood eosinopenia induced by glucocorticoids could be partly due to various metabolites discharged into the circulation following tissue action of these hormones and that electrolytic equilibrium of the blood also plays a role in the response of the eosinophil to cortisone. The increase in gamma globulin seen in eosinopenia may coincide with other nitrogenous and carbohydrate metabolites, which, like albumin and globulin, act upon eosinophils directly and indirectly through the hypophyso-adrenal cortical system (Olson, 1968).

Eosinopenia has been shown to exist following stress (Simpson, 1967). Seyle (1949) states that eosinopenia is a characteristic feature of the alarm reaction. Trauma also produces a generalized peripheral eosinopenia. Surgical operations initate an increased production of glucocorticoids by the adrenal cortex resulting in a profound drop in the eosinophils, the greatest drop being the first day post-operatively followed by a gradual return to normal (Coppinger and Goldner, 1950).

According to Archer (1963) local stress causes a local release of histamine and then through diffusion histamine is released into the peripheral circulation. The increased histamine stimulates the anterior pituitary lobe to release ACTH which in turn causes release of adrenal corticoids. The increase of corticoids inhibits

the biogenesis of histamine reducing the circulating histamine below normal level. Thus the lowered circulating histamine inhibits the production and release of eosinophils from the bone marrow resulting in the subsequent peripheral eosinopenia.

In Honsinger's (1972) view, a reduction in circulating eosinophils can be produced consistently by any of several procedures, but because of the interrelated variables of production, tissue localization, destruction and possibly others, the development of any fundamental understanding of the mechanisms involved is grossly hindered.

The eosinopenic effect of physical and emotional stress has always been attributed to the release of adrenocortical hormones and catecholamines and the measurement of the eosinopenic effect of stress of exercise has been proposed as a simple screening device for estimation of adrenal function (Renold et al., 1951). Whether this can be reproduced in adrenalectomized humans has not been determined but may be related to the eosinopenia induced by high atlitude (Honsinger, 1972).

F. EXERCISE STRESS AND EOSINOPHIL RESPONSE

The use of eosinophil counts to indicate acute exercise stress of fatigue have been limited. Weber (1971) concluded that:

> "Eosinopenia assumes a prominent position in explaining Maximum Voluntary Performance as it accounts for approximately one-half of the net per cent contribution of independent variables to the explained variance when

different run treatments are combined",

and

"The per cent eosinophil reduction does significantly discriminate at the 1% level between different workloads of treadmill running which are at least four minutes apart."

Weswig (1974) noted from his study of swimmers in training that after swimming 5,500 meters an increase in the white blood cells was found which was consistant each of four months of training, concluding that the increase was due to the stress of exercise.

De Lanne et al (1960) found that the total leucocyte count is influenced by duration and intensity of exercise, and that changes were significantly greater in females than in males. Also, the eosinophils increased early during the exercise and their number remained elevated during submaximal work and for five minutes after maximal work. One hour later they were below resting level. The men had significantly more eosinophils than the women at rest and this difference persisted during exercise. Because of the small proportion of eosinophils compared with other types of cells and the variability among individuals, the relative number of eosinophils was not significantly altered by exercise or heat stress; but the total number of leucocytes was significantly modified by both exercise and heat stress. These authors conclude that in certain types of stress the level of cortical steroids in the peripheral blood has been shown to rise resulting in the decreased number of circulating eosinophils so that eosinopenia may be interpreted as

being a reflection of the compensatory hormonal adaptations.

Karpovich (1971) notes that both the mechanism and the significance of exercise leucocytosis are not well understood but that there is general agreement that this increase is due to "washing out" of the white blood cells from the storage places (bone marrow, spleen, liver, lungs) caused by the greatly increased circulation. Also, that the lactic acid content of the blood, blood sugar and blood pressure have no separate relations to exercise leucocytosis, and excitement alone has no effect upon leucocytosis.

Treloar (1977) investigated the stress of handling and taking blood samples from adult male Sprague Dawley rats every 10 minutes for 60 minutes and found eosinophenia to occur within 10 minutes. The fall in eosinophils reached a low of 63% of the non-stressed sample by 40 minutes. He found that this eosinophenia was mediated by way of the adrenocortical steroids as indicated by the failure of both adrenalectomized and hypophysectomized rats to react to the stress of handling and sampling with an eosinopenic response similar to the intact controls. He concluded that the response was not dependent on the stimulation of beta-adrenegic receptors by epinephrine since propranolol, a beta-adrenergenic blocking agent, failed to produce any diminution of the eosinophenic response.

Frenkl (1969) found that the rise in steriod concentration in response to muscular exercise is less marked and of considerably shorter duration in trained than in untrained subjects; this difference cannot be attributed merely to differences in the rate of steroid metabolism. He concluded that the difference in the

stress reaction between trained and untrained organisms seems to be due to the smaller and less sustained rise in ACTH secretion in trained subjects.

This difference in the rate of disappearance of steriods after exercise may be due to enhanced steroid metabolization in the trained organism, or to an increased capacity of the tissues to bind these hormones.

Keeney (1960) found that a period of physical training increases the functional capacity of the adrenal cortex of rats and that this increased capacity is dependent upon continued training since it is lost after a few days of reduced physical activity. This compares favourably with the observations of Berger (1954) who found that trained athletes exhibited a greater and more prolonged eosinophil response to a given work load and to the injection of epinephrine than did a group of sedentary controls.

Sealander and Guess (1970) found that the magnitude of the eosinophil response to swimming (in rats) was not correlated with the amount of stress as measured by swimming time, water temperature, age, weight or sex.

Frenkel (1966) suggests that either the rate of steroid disappearance from the blood is increased or that the stage of resistance is maintained not by cortical steroids but by some other humoral factor ("resistin") to account for the fact that during the stage of resistance in adaptation to exercise the steroid level of blood is normal.

Speirs and Meyer (1949) found that a mild stress produces a great decrease in the number of circulating eosinophils which lasts over a period of eight hours. Following this there is an increase in the number of eosinophils and a return to normal within approximately sixteen hours after initial stimulation. A second dose of the same stress was found to be cumulative in that after eight hours the number of eosinophils still showed a decrease of 96%, as compared with a single dose group which returned to normal levels after eight hours.

Suzuki et al (1967) conducted an experiment to determine the effect of muscular exercise on adrenocortical secretion in dogs. The dogs were trained at various speeds and distances to provide slight to exhausting exercise conditions, after which venous blood was analyzed. The authors found that only exhaustive exercise provoked marked increases in adrenocortical function thus concluding that cortical secretion was not related to exercise, its duration or intensity but simply to exhaustion.

Steadman and Sharkey (1969) conducted a study to determine the effects of a strenuous but non-exhausting program of physical exercise on adrenocortical activity and used five male volunteers from the physical education service program. The four weeks of training consisted of a modified Balke test (T180) on Monday and Wednesday and a progressive treadmill walk to exhaustion on Friday. Seventeen-keto-steroids (17-KS) and seventeen-ketogenic steroids (17-KGS) were estimated from twenty-four hour urine collections. This was proceeded by one week of control where samples were

collected on the Tuesday and Thursday. Circulating eosinophil counts were determined on three subjects immediately following the T180 walks (Monday and Wednesday). Their results showed that despite a physiologically adjusted (increased) work load throughout the four weeks of training, the early increases of 17-KGS and decreases in both 17-KS and eosinophil levels returned to near normal (control) levels. Unfortunately with only five subjects, and only three on eosinophil counts, the large variability in their data does not permit confident interpretation. The authors emphasized the influence of other uncontrolled factors which are known to affect adrenocortical activity such as heat, cold, altitude and a variety of emotional situations. They felt that the inability to control the other possible stressors undoubtedly accounted for some of the variability so evident in the study. Apparently the length of exposure to the exercise situation did not allow adequate familiarity and eventual reduction of the emotional response to the exercise task as the authors had hoped.

Despite the problems associated with this particular study and even though the early drop in eosinophil count was consistant with previous studies, Steadman and Sharkey felt that the use of the eosinophil count as an index of stress was "in question." This was not based on their own results but on the fact that epinephrine has been shown to induce eosinopenia in the absence of functioning adrenocortical tissue.

Steadman and Sharkey go on to suggest from their results and others (e.g. Susuki, 1967) that exercise, by itself, does not

elicit increased adrenocortical activity and that the emotional response to the task may be more important in this regard than the work itself. They further suggest that because the exercise loads were adjusted to meet any adaptation to the training load, presumably based on heart rate response to the treadmill stress, the return to normal adrenal synthesis and secretion activity during the last weeks of training was due to adrenal exhaustion and not adaptation. Frenkl et al (1968) present conflicting findings when they compared trained and untrained rats. They observed that the response to exogenous ACTH was essentially identical in trained and untrained rats which indicates that the difference revealed in previous (1966 and 1962) experiments for corticosterone levels was due to adaptation of the pituitary-adrenal axis, and not to exhaustion of adrenocortical hormone production. They conclude by stating that "it seems that adaptation of endocrine functions to regular exertion is the result of a multifold adjustment at different levels: ACTH is not mobilized readily, consequently the adrenocortical response is diminished, the rate of elimination of steroid hormones is augmented. The observed response is the result of these complex adjustments." Contrast this with Steadman's et al conclusion that "the lowered 17-KS levels in the early weeks of training may have been due to increased demands for the precursor substance cholesterol. Eosinopenia may have been due to either adrenocortical or adrenomedullary causes. Returns toward control levels were interpreted as signs of familiarization with the demands of the exercise....the exercise, by itself did not increase adrenocortical

activity."

Chin and Evonuk (1971) found a significant decrease in plasma corticosterone concentrations between exhaustively exercised rats and the control and moderately exercised groups. They felt that their results were in accord with those of Frenkl and Csalay (1962) and quote this source as hypothesizing that "an approaching adrenal exhaustion caused the corticosterone levels to decrease below normal concentrations." It is interesting to note that Steadman and Sharkey quote from the same source (Frenkl and Csalay, 1962) and then to compare what Frenkl and Csalay had to say in 1968, quoted above.

CHAPTER III

METHODS AND PROCEDURES

A. SUBJECTS AND GROUPINGS

Twenty-nine male swimmers from a competitive swim club (The Olympians) volunteered as subjects for this study. The average age was fourteen years and seven months. Table II details some of the subjects' characteristics including pre-training values for total white blood cell counts (TWBC) averaged over the two weeks prior to training, maximum oxygen consumption (MVO_2), percent body fat (%BF) and the average eosinophil (EOS) levels for the two weeks prior to training which represent the basal levels for these subjects.

All subjects were competent in all four competitive strokes with only a few of the youngest members having less than two years of competitive swimming experience. The subjects were assigned to groups as follows:

Group One: High Intensity/High Total Work
Group Two: High Intensity/Low Total Work
Group Three: Low Intensity/High Total Work
Group Four: Low Intensity/Low Total Work

TABLE II CHARACTERISTICS OF SUBJECTS

	SUBJECT	MVO_2 (ml/kg/min) PRE	MVO_2 (ml/kg/min) POST	MEAN BASAL TWBC cells x 10^3 / mm^3	MEAN BASAL EOS cells x 10^3 / mm^3	% BF PRE	% BF POST
GROUP 1 (H/H) n = 8	1. JK	66.4	64.6	4.94	0.215	8.9	8.2
	2. BS	63.4	62.5	5.00	0.110	16.0	16.5
	3. SS	70.3	64.3	7.62	0.084	9.6	10.3
	4. BL	63.6	62.1	7.20	0.348	12.2	11.5
	5. DD	69.5	66.2	5.97	0.254	10.2	10.5
	6. RB	67.5	65.0	5.40	0.403	18.5	18.7
	7. SM^C	58.2	56.6	6.38	0.178	7.4	8.2
	8. BL	66.2	63.5	5.30	0.368	5.5	5.6
GROUP 2 (H/L) n = 7	9. WM	65.4	58.6	6.19	0.010	8.6	8.2
	10. KK	63.6	61.9	4.92	0.153	17.7	17.0
	11. CR	54.0	60.4	6.30	0.288	16.4	13.5
	12. TS	59.2	64.5	7.45	0.228	13.3	12.8
	13. RB	67.5	69.9	5.61	0.443	15.4	16.1
	14. JD	69.0	69.7	6.80	0.252	12.9	10.2
	15. GB	72.2	68.0	6.23	0.254	11.2	11.8
GROUP 3 (L/H) n = 7	16. JA	76.9	67.0	5.80	0.196	5.2	5.3
	17. SF	62.8	62.5	9.78	0.266	18.6	17.2
	18. PL	60.6	65.8	5.99	0.158	10.4	10.2
	19. SS	68.7	64.7	6.52	0.198	6.5	6.2
	20. PL	67.8	68.2	5.24	0.205	5.4	5.7
	21. CM	64.8	62.6	6.63	0.218	14.7	13.2
	22. ST	60.1	62.4	4.53	0.103	12.6	12.2
GROUP 4 (L/L) n = 7	23. TS	64.8	56.2	6.11	0.080	15.1	13.6
	24. GB	66.8	68.3	4.74	0.235	14.5	15.1
	25. DA	56.5	60.2	4.65	0.103	14.4	14.6
	26. RW	61.0	53.3	6.04	0.596	19.0	19.5
	27. JS	61.0	54.5	6.48	0.435	12.9	13.6
	28. SM	68.8	67.9	5.76	0.160	10.9	11.1
	29. RS	57.9	61.6	5.71	0.160	16.1	17.0

These groups were all used initially as control groups in order to establish basal levels for TWBC and EOS during the two weeks prior to training. For the six-week training period which followed, the members of each group were assigned the appropriate work (exercise stress) for their group.

A one-way ANOVA was used to determine if there were any initial significant differences between the groups on the following parameters: TWBC, EOS and MVO_2. Appendix B shows that there is no significant differences between the groups on TWBC, EOS levels or MVO_2 and %BF estimations.

TableIII shows the experimental design used in this study. There were 7 or 8 subjects assigned to each of the four groups described above.

TABLE III SCHEMATIC REPRESENTATION OF THE EXPERIMENTAL DESIGN

TOTAL WORK	HIGH (15)				LOW (14)			
INTENSITY	HIGH (8)		LOW (7)		HIGH (7)		LOW (7)	
TESTS*	PRE	POST	PRE	POST	PRE	POST	PRE	POST

* Refers to PRE and POST exercise and PRE and POST training values.

B. TRAINING STRESS

The training stress was designed to simulate as closely as possible the actual training situation while maintaining some control of the variables which influence the adaptation to training. After the two weeks of basal sample collection during which time no training was done, the subjects began a six-week training regimen which closely resembled previous start-of-season workouts. Initially, the training was light and only one session per day was required to complete the work assigned. Gradually, the sessions increased in terms of the distance covered until two sessions per day were needed to complete the planned workout.

All subjects, irrespective of group, began with the same total work (2000 yards) and at the same intensity (personal best time plus 30%). As the workouts progressed, the intensity quickly changed so that the high intensity groups were working at a level equivalent to their personal best time plus 15% additional time (PBS+15) whereas the low intensity groups trained at PBS+25. The total work was also gradually increased each day for all groups but more rapidly for the High Total Work groups (groups 1 and 3) than for the Low Total Work groups. Graph 1 illustrates the application of the training stress in terms of the distance in yards swum per session. Initially large increases were made in distance swum for all groups but these were gradually reduced in magnitude in the succeeding weeks.

Rest periods were controlled only to the extent that the High Intensity groups enjoyed less rest than the Low Intensity groups, but no definite ratio was prescribed due to the difficulty of monitoring both repeat times and rest times for all members of all groups.

C. THE BLOOD SAMPLES

During each week of the study, blood samples were collected by venipuncture on Monday, Wednesday and Friday by trained personnel. For the two weeks prior to training, the samples were collected between 7:00 and 7:30 a.m. When the training period began, twenty randomly selected subjects, five from each group gave pre-exercise samples and all subjects gave post-exercise samples. The former were drawn between 5:40 and 6:00 a.m. while post-exercise samples were done between 7:30 and 8:00 a.m.

The samples were collected in vacutainers which contained the anti-coagulent EDTA and were processed in the laboratory within the required forty-eight hours.

D. ANALYSIS OF THE SAMPLES

All blood samples were analysed on the Technicon Hemalog-D Analyzer. In the initial stages, samples were also run on the Hemalog-8 analyzer to obtain values for hemoglobin, red blood cell counts, white blood cell counts, hematocrit and mean corpuscular volume. The Hemalog-D obtained values for total WBC and a complete differential count which includes the eosinophil levels for each sample processed. See Appendix C for additional information on the Hemalog-D analyzer.

E. STATISTICAL TREATMENT

As mentioned previously, a one-way ANOVA was used to detect any initial significant differences between the groups prior to training on the variables TWBC, EOS, MVO_2 and %BF. Further analysis of variance techniques were used to show any significant differences between groups on TWBC or EOS for comparison of pre and post-exercise values (to gauge the effects of acute exercise); comparison of groups on pre-exercise values (to determine if there were chronic effects due to the training prescribed); and, a comparison of groups on post-exercise values alone (to see if there are group differences due to the intensity or the amount of work done or an interaction of both).

The above statistical treatment was performed using a package computer program called SPSS "ANOVA". Post hoc procedures involved the use of a Scheffe multiple-comparison test. Significant differences were accepted at the alpha level p is less than 0.05 where p is the probability that no difference exists between means.

The SPSS "ANOVA" program is specifically designed to carry out analysis of variance for the unequal N case with repeated measures on Factor C.

F. CRITERION TESTS

To provide a basis for comparison of pre and post training levels, three criterion tests were used.

Firstly, MVO_2 values were obtained using the treadmill and the Beckman Metabolic Cart. All subjects were given a pre and post-training evaluation test which involved a five minute warm-up and orientation to

the treadmill and other required apparatus. Following a five minute rest period, the subjects were run to exhaustion on the treadmill with the increasing load being applied via increases in speed and grade according to Astrand (1970); with the exception that the continuous work period of five to eight minutes was attained using 2.67% increases in grade every two minutes.

At each test the body weight was measured prior to the run with the subject clad only in swim briefs. Cardiometer leads were attached for each test in order to monitor directly the heart rate response to the workload. The subject ran until MVO_2 had been reached. The criterion that MVO_2 had been reached was a levelling off of VO_2; less than 150 ml/min increase over the previous workload (Astrand, 1970).

During all MVO_2 evaluation tests, the subject was fitted with a nose clip, a rubber mouth piece and a Rudolph valve. The expired air was analyzed automatically every 30 seconds by the Metabolic Measurement Cart (Beckman) which was calibrated prior to each test.

A second criterion test was a series of maximal swims(MS) over 100, 200 and 400 yards, using the swimmers preferred stroke. The difference between pre and post training values was used as a criterion of improvement. The mean difference for each group was used to compare the degree of improvement.

A third test was that of percent body fat (%BF) which was also performed prior to the start of training and in the last week of training. This test utilized the standard densitometry procedures for determining %BF in the tank and, again, means for each group were compared. The recorder was calibrated prior to each testing session.

With the above tests, one is able to determine if the training that each of the groups experienced was, in fact, vigorous enough to elicit improvements/changes in the parameters measured. If so, then those conditions should also produce changes in TWBC and EOS counts if any changes are to occur at all.

G. QUESTIONNAIRE

An "Activities Questionnaire" was given to each member of all groups requiring information on the level and nature of the subjects' activities in the six weeks prior to the beginning of the study. (See Appendix D.) The object was to ascertain the type of activity the majority of subjects had been accustomed to during the preceeding weeks. This was done because some researchers have found that swimmers show higher MVO_2 values for tests performed while swimming in a flume than for tests performed on a bicycle ergometer or a treadmill. The logic being that the specificity of the test on the treadmill makes it less than ideal for the evaluation of swimmers.

CHAPTER IV

RESULTS

No statistical difference was shown to exist on the basal values for TWBC or EOS between the groups. The Scheffe multiple-comparison test previously mentioned, indicated that the Type 1 error was not, in fact, made thus the groups were very similar when compared in initial levels of TWBC and EOS.

When all pre-exercise samples where pooled for the six weeks of training, a one-way ANOVA on the dependent variable EOS indicated a significant difference between Group 4 and Groups 1, 2 and 3. The Scheffe test showed that at the 0.05 level, the Type 1 error was not made. There was no significant differences between the groups on the TWBC counts.

Using a one-way ANOVA to examine the pooled post-exercise samples there was found to be a highly significant difference between Groups 1 & 3, 2 & 4 and 3 & 4 according to the Scheffe test. Again, there was no significant difference between groups on the TWBC counts for the pooled post-exercise samples.

There was also a significant conditions effect due to the total work performed and a significant interaction's effect between intensity and total work on EOS counts when a two-way ANOVA was used on pooled pre-exercise data. There was a similar response indicated on the pooled post-exercise data in that there was a highly significant interactions effect between intensity and total work but there was no main effect differences for eosinophils. There were no significant differences at all

for TWBC on pooled pre or post-exercise data.

A two-way ANOVA on pooled pre-exercise samples showed highly significant differences between groups on EOS. This difference was again attributed to the amount of work performed (total work) rather than the intensity of the effort. There was also a highly significant interactions effect on the EOS count between intensity and total work performed. The TWBC, however, showed a difference, for the first time, between the groups on the pre-exercise samples. This was due to the intensity of effort performed rather than total work but this difference was not significant at the 0.05 level ($F = 2.949$ and $F' = 3.06$).

A three way ANOVA on the EOS count indicated a highly significant main effect due to the pre-post-exercise condition and to the total work performed, but not to the intensity of the work performed. There was also a highly significant two-way interactions effect between intensity and total work performed but no other interactions were significant. None of the variables of intensity, total work performed or pre/post exercise conditions had a significant effect on TWBC.

Graph 2 shows a graph representing the EOS post-exercise counts for each group for the duration of the study while Graph 3 shows the TWBC post-exercise counts for the same period. Graphs 4 and 5 show EOS and TWBC, respectively, for pre-exercise blood samples, whereas Graphs 2 and 3 are for post-exercise samples.

It should be noted that samples taken on days 1 - 6 (all of the

basal samples), 7 - 10 and 18 (training samples) were analyzed on a Hemalog-D at the Royal Alexandria Hospital in Edmonton. Samples taken on days 11 to 17 were shipped to the University Hospital in Saskatoon for analysis on another Hemalog-D due to technical problems with the first machine.

The results of the activities questionnaire showed that 20 of the 29 subjects engaged in some form of activity other than swimming during the "off-season" period of two months, mostly at a fairly high level (i.e. "4 sessions per week or more"); these activities included cycling, volleyball, cross-country running, track and field, soccer and some swimming. The remainder either did nothing on a regular basis or similar activities at only low or moderate levels (i.e. one or two sessions per week).

The criterion tests, MVO_2 and %BF showed no significant differences between groups by a three-way ANOVA of group means on pre and post-training values over the six weeks of training.

CHAPTER V

DISCUSSION

From the preceding results one may see that firstly there seems to be a chronic eosinophilic response to the stress of training which is evident in all groups (See Graph 4). Group 4 has an overall milder response to the training whereas Group 3 seems to have the greatest response with Groups 1 and 2 fluctuating in between. This graph illustrates the highly significant differences found between the groups on EOS count: $1 \neq 3$, $2 \neq 4$, $3 \neq 4$ (Scheffe $p=0.05$). Conversely, there was no significant differences found between Groups 2 and 3, Groups 2 and 1 or 1 and 4. The gradual eosinopenia seems to be in direct relationship with the gradual increase in the work done per session (in terms of distance swum). This is in accordance with the findings of Keeney (1960) who suggested that a period of physical training increases the functional capacity of the adrenal cortex which is concomitant with the well-known enlargement of the adrenal gland due to training.

As the statistical analysis showed, there are no significant differences between the groups on TWBC counts and this is illustrated by Graph 3, which indicates no relationship between the increasing stress of training and the TWBC response. This contrasts with the findings of De Lanne (1960) who claims that the reactions of the white blood cells depend upon the duration and intensity of the exercise, the individual's fitness level and the recovery time prior to sampling time. In the present study recovery time prior to sampling varied randomly but all samples

were collected within 30 minutes of each other while the groups were similar on fitness levels as defined by MVO_2 values; and no differences were found on WBC counts.

According to the statistical analysis performed on the EOS, the differences between the groups was due to either the total work performed, or perhaps, more accurately, the duration of the exercise bout rather than the intensity of the efforts during training. This seems to be indicated by the pattern of differences between groups 1 & 3, 2 & 4, and 3 & 4 whereas groups 2 & 3, 2 & 1 and 1 & 4 have no such differences. That is, those groups who differ most in the time taken to complete the workout also differ significantly on the EOS count.

Again, there is no such response with the total WBC parameter. This is in direct conflict with the previous findings by De Lanne (1960) who suggests that the number of eosinophils is not altered by exercise stress because of the variability among individuals and the small proportion of eosinophils compared with other types of cells but that the total WBC were significantly modified by both exercise and heat stress.

On the other hand Bilger (1954) found that trained athletes exhibited a greater and more prolonged eosinophil response to a given work load than did sedentary controls. Keeney (1960) found that the increased capacity of the adrenal cortex of rats following physical training was dependent upon continued training since it is lost after a few days of reduced physical activity. But Sealander and Guess (1970) concluded from their research that the magnitude of the eosinophil response to swimming (in rats) was not correlated with the amount of stress as measured by swimming time (i.e. duration), water temperature, age, weight, or sex.

Steadman and Sharkey (1969) conducted a study to determine the

effect of a strenuous but non-exhausting program of physical exercise on adrenocortical activity. Their results showed that despite a physiologically adjusted (increased) work load throughout the period of four weeks of training, the early increases of 17-KGS and decreases in both 17-KS and EOS, levels returned to near normal (control) levels. This contrasts with the present findings that EOS levels returned to near normal values only after six weeks for groups 1, 2 and 3 whereas group 4 stayed at near normal values throughout.

As indicated previously there were significant pre and post-exercise differences for both EOS and TWBC but only the former was significant. Again the differences between pre and post-exercise values was due to the duration (or total work performed) and not to the intensity of the exercise session.

Previous research (Magel et al, 1975; Homer et al, 1972 & 74;Dixon and Faulkner, 1971; Shepard et al, 1974; McArdle et al, 1971; Miyashita et al, 1970 and Kipke, L. 1974) has indicated that differences do exist in evaluating MVO_2 values for athletes. These differences depend upon the mode of training and the testing mode; that is, treadmill, bicycle ergometer or swimming flume. The specificity of the training and the test alike create significant differences which make some comparisons meaningless. With this in mind, it is not surprising that there was no significant differences found between pre and post-training values for MVO_2 in this study.

The high values found in some of the subjects coincided with high activity levels in sports such as soccer, cycling and cross-country running which have obvious and close relationship to the treadmill test for MVO_2 used in this study. During the course of the swimming training and

the subsequent exclusion of other activities entirely there was an immediate shift in the emphasis of demand from the legs to the arms. Thus the post-training evaluation of MVO_2 largely reflected this drop in values especially in those subjects with extremely high pre-training levels ($\geq$ 70 ml/kg/min). At the same time, those with fairly low MVO_2 values at the pre-training evaluation test (mostly due to low levels of "off-season" activity) managed to increase markedly their post-training values.

The %BF showed similar responses across the groups of subjects but for slightly different reasons. Both the type of activity indulged in during the off-season and the change of season from summer to winter, influenced the maintenance of or increase in %BF values in the majority of subjects; very few lost weight due to decreases in body fat. It is commonly accepted (Astrand, 1970) that swimmers have normally, a higher per cent of body fat than other endurance athletes; this is usually attributed to the need to maintain body temperatures in water.

The eosinophil response to the exercise stress seems to reflect fairly accurately the functional involvement of the adrenal cortex: the greater the total work performed, the greater is the output of corticosteroids which in turn decrease the eosinophil levels markedly. As the work increases (distance swum) so the EOS drops; there is no general return to normal values for groups 1, 2 and 3. Group 4 generally shows a downward trend but only slightly. Groups 1 and 2 alternate positions of order throughout, showing a downward trend until sampling day 15 (See Graph 2.). Group 3 is consistently lower throughout the series of samplings but also begins an upward trend after the 15th sampling day. This point represents the start of the 5th week of training and this change in the direction of eosinophil response may be interpreted as

being the point where adaptation is occuring on a large enough scale to be monitored by the eosinophil count.

De Lanne (1960) suggests that there are significant changes in TWBC due to muscular activity and that these reactions depend upon the duration and intensity of the exercise, the individuals' fitness level and the recovery time prior to sampling. Although no statistically significant changes were detected in the present study, there was some indication that intensity had an effect on TWBC.

From the foregoing discussion, it can be seen that the EOS count may be used to indicate a state of "overtraining" i.e. where increases in work or even continuation at the present level brings no adaptation. This is possible provided that a true basal value for the count has been established as a reference point prior to the start of the training season. If the individual takes part in other sports quite actively, during the "off-season", a true basal level may not be possible. Thus when the EOS count has dropped drastically during a fairly heavy period of training, it is feasible to control the failing adaptation by reducing or at least levelling off the amount of training (in the form of yards swum or total work performed) that is required.

The TWBC seems to be little affected by either the amount of work performed or the intensity with which it is done. So there seems to be little benefit in following this parameter solely.

Regarding methodology of this study, perhaps a more vigorous approach which controls the intensity of the workout more closely (especially the rest periods between efforts and sets of efforts) may yield significant results from the variable of "intensity".

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

SUMMARY

Much of the results of the study point to both a chronic and an acute eosinophil response to the stress of training. The chronic response being illustrated well in Graph 4 by the general gradual drop in EOS levels for groups 1, 2 & 3; group 4 remains pretty much at basal levels throughout. At the end of the six-week training period, the downward trend seems to have levelled off, at least, and is possibly an indication of an upward, recovery trend. This latter segment may well be interpreted as the adaptation period reported by Keeney (1976) and Frenkl and Csalay (1962). Meanwhile the acute response to the stress of training is of significantly greater magnitude over the six-week period as illustrated in Graph 2. But these values also show a trend towards basal levels in the last week (3 sampling days) of the training period. Evidently, adaptation is beginning to occur with the normalization of the eosinophil response.

According to the statistical analysis, the factors which contribute to the eosinophil response are mainly those of duration and/or total work performed. This same analysis indicates that there are significant differences between the groups on the magnitude of response to exercise stress between pre and post-exercise values. Also quite distinctly

shown, was the lack of significant difference between the groups on the TWBC values, even pre and post-exercise. Despite this, there can be seen in Graph 3, a very slight increase in the counts for all groups, generally, on post-exercise values over and above the pre-exercise (or chronic) levels indicated in Graph 5. This may suggest at least a trend which is not as responsive as the EOS count.

It is suggested that intensity did not show up as a significant factor because of the lack of adequate control of the rest periods between efforts during training. This was due to the practical problem of monitoring every facet of training for so many subjects, simultaneously. Also, the fact that the intensity remained much the same througout the six weeks of training, may indicate that adaptation had occurred at that level and was no longer a significant factor.

CONCLUSIONS

The following points may be concluded from this study:

1. There is both an acute and a chronic eosinopenia resulting from the stress of swimming training over a period of six weeks.

2. Adaptation to the stress of training seems to occur at approximately the fifth week of gradually increasing (but at a deminishing rate) total work output, in terms of yards swum.

3. The TWBC response to exercise stress over a period of six weeks is far less evident than the EOS response.

4. With basal values for comparison, the EOS response to the stress of exercise reflects fairly accurately the level of adaptation of the body even over just six weeks of training.

RECOMMENDATIONS

1. Due to the exploratative, qualitative nature of this study some caution is required in interpreting the findings. Until further more quantitative evaluations of the eosinophil response to exercise stress can be made these present results should be seen as trends only. More stringent controls approaching those used in a laboratory situation are required before precise estimates can be made in terms of how much response to 'x' amount of exercise stress.

2. A closer look at the role of intensity in the eosinophil response is also needed. It may well be that intensity, like total work and/or duration, needs to be increased gradually but continually in order that an eosinophil response be measured.

3. With greater controls over such variables as the time lag between the completion of exercise and the sampling of blood one may find less variance in values obtained.

4. The use of fewer subjects at one time would enhance the control over many of the variables such as; activity during pre-training periods, the actual amount of work done per training session, the intensity of the efforts by perhaps using heart rate monitors instead of times which are absolute, not relative, values.

PRACTICAL IMPLICATIONS

Despite the fact that the present findings are positive with respect to the use of the eosinophil count as an indicator of stress the practical use of this index has some limitation due to the technique of drawing samples and the analysis required for precision thereafter. Unless the coach has ready access to the Hemalog-D or a medical centre which can process the samples using a similar machine, then there is no real value in following this parameter.

Basal values must also be well established prior to the commencement of the season and influencing variables such as emotional factors must also be taken into consideration.

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APPENDICES

APPENDIX 'A'

STATEMENT OF HYPOTHESES

THE HYPOTHESIS

Generally stated the hypothesis of this study was that the eosinophil response to Exercise stress varied according to the intensity and the amount of total work completed and that the chronic response to exercise stress was a better index of adaptation that the acute response.

In scientific notation the hypothesis would be stated as follows:

$$Ho: \bar{X}_1 = \bar{X}_2 = \bar{X}_3 = \bar{X}_4 \quad \text{(null hypothesis)}$$

alternatively,

$$H_I: \neq \bar{X}_1 \neq \bar{X}_2 \neq \bar{X}_3 \neq \bar{X}_4 \quad \text{(alternate hypothesis)}$$

APPENDIX 'B'

SUMMARY OF STATISTICAL ANALYSIS

ONE - WAY ANOVA ON BASAL EOSINOPHIL LEVELS

ANALYSIS OF BLOOD SAMPLES
ONE-WAY ANOVA ON BASAL MVO2 %BF EOS & TWBC 05/20/79 PAGE 6
FILE NONAME (CREATION DATE = 05/20/79)

ONEWAY

VARIABLE EOS

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	0.0155	0.0052	0.294	0.8295
WITHIN GROUPS	25	0.4407	0.0176		
TOTAL	28	0.4562			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	8	0.2450	0.1197	0.0423	0.0840	0.4030	0.1449 TO 0.3451
GRP2	7	0.2326	0.1317	0.0498	0.0100	0.4430	0.1108 TO 0.3544
GRP3	7	0.1920	0.0507	0.0192	0.1030	0.2660	0.1451 TO 0.2389
GRP4	7	0.2527	0.1918	0.0725	0.0800	0.5960	0.0753 TO 0.4301
TOTAL	29	0.2311	0.1276	0.0237	0.0100	0.5960	0.1825 TO 0.2796
FIXED EFFECTS MODEL		0.1328	0.0247				0.1803 TO 0.2818
RANDOM EFFECTS MODEL		0.0493	0.0247				0.1526 TO 0.3095

WARNING - BETWEEN COMPONENT VARIANCE IS NEGATIVE
IT WAS REPLACED BY 0.0 IN COMPUTING ABOVE RANDOM EFFECTS MEASURES

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE -0.0017

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.5180, P = 0.075 (APPROX.)
BARTLETT-BOX F = 2.721, P = 0.043
MAXIMUM VARIANCE / MINIMUM VARIANCE = 14.293

ONE-WAY ANOVA ON BASAL TOTAL WHITE BLOOD CELL LEVELS

ANALYSIS OF BLOOD SAMPLES
ONE-WAY ANOVA ON BASAL MVO2 %BF EOS & TWBC
FILE NONAME (CREATION DATE = 05/20/79)

05/20/79 PAGE 4

- O N E W A Y -

VARIABLE TWBC

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	2.0529	0.6843	0.554	0.6502
WITHIN GROUPS	25	30.8747	1.2350		
TOTAL	28	32.9276			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	8	5.9775	1.0150	0.3589	4.9400	7.6300	5.1289 TO 6.8261
GRP2	7	6.2143	0.8081	0.3054	4.9200	7.4500	5.4669 TO 6.9617
GRP3	7	6.3557	1.6757	0.6334	4.5300	9.7800	4.8059 TO 7.9055
GRP4	7	5.6414	0.6947	0.2626	4.6500	6.4800	4.9989 TO 6.2839
TOTAL	29	6.0448	1.0844	0.2014	4.5300	9.7800	5.6323 TO 6.4573
FIXED EFFECTS MODEL			1.1113	0.2064			5.6198 TO 6.4698
RANDOM EFFECTS MODEL			0.4127	0.2064			5.3881 TO 6.7016

WARNING - BETWEEN COMPONENT VARIANCE IS NEGATIVE
IT WAS REPLACED BY 0.0 IN COMPUTING ABOVE RANDOM EFFECTS MEASURES

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE -0.0760

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.5645, P = 0.034 (APPROX.)
BARTLETT-BOX F = 1.816, P = 0.142
MAXIMUM VARIANCE / MINIMUM VARIANCE = 5.818

ONE-WAY ANOVA ON MAXIMAL OXYGEN CONSUMPTION (PRE-TRAINING)

ANALYSIS OF BLOOD SAMPLES 05/20/79 PAGE 2
ONE-WAY ANOVA ON BASAL MVO2 %BF EOS & TWBC
FILE NONAME (CREATION DATE = 05/20/79)

- O N E W A Y -

VARIABLE MVO1

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	55.5806	18.5269	0.698	0.5623
WITHIN GROUPS	25	663.9827	26.5593		
TOTAL	28	719.5632			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	8	65.6375	3.8822	1.3726	58.2000	70.3000	62.3919 TO 68.8830
GRP2	7	64.4143	6.1705	2.3322	54.0000	72.2000	58.7076 TO 70.1210
GRP3	7	65.9570	5.8449	2.2092	60.1000	76.9000	60.5515 TO 71.3626
GRP4	7	62.3999	4.5654	1.7256	56.5000	68.8000	58.1776 TO 66.6222
TOTAL	29	64.6379	5.0694	0.9414	54.0000	76.9000	62.7096 TO 66.5662
	FIXED EFFECTS MODEL		5.1536	0.9570			62.6669 TO 66.6088
	RANDOM EFFECTS MODEL		1.9140	0.9570			61.5923 TO 67.6834

WARNING - BETWEEN COMPONENT VARIANCE IS NEGATIVE.
IT WAS REPLACED BY 0.0 IN COMPUTING ABOVE RANDOM EFFECTS MEASURES

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE -1.1092

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.3520, P = 0.589 (APPROX.)
BARTLETT-BOX F = 0.545, P = 0.651
MAXIMUM VARIANCE / MINIMUM VARIANCE = 2.526

ONE-WAY ANOVA ON PERCENT BODY FAT (PRE-TRAINING)

ANALYSIS OF BLOOD SAMPLES 05/20/79 PAGE 8
ONE-WAY ANOVA ON BASAL MVO2 %BF EOS & TWBC
FILE NONAME (CREATION DATE = 05/20/79)

- O N E W A Y -

VARIABLE %BF1

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	88.3367	29.4455	1.896	0.1561
WITHIN GROUPS	25	388.3040	15.5322		
TOTAL	28	476.6406			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	8	11.0375	4.3572	1.5405	5.5000	18.5000	7.3948 TO 14.6802
GRP2	7	13.6428	3.1416	1.1874	8.6000	17.7000	10.7374 TO 16.5483
GRP3	7	10.4857	5.1278	1.9381	5.2000	18.6000	5.7433 TO 15.2281
GRP4	7	14.7000	2.5305	0.9564	10.9000	19.0000	12.3597 TO 17.0403
TOTAL	29	12.4172	4.1259	0.7662	5.2000	19.0000	10.8478 TO 13.9866
FIXED EFFECTS MODEL			3.9411	0.7318			10.9100 TO 13.9245
RANDOM EFFECTS MODEL			2.0176	1.0088			9.2069 TO 15.6276

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 1.9214

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.4272, P = 0.260 (APPROX.)
BARTLETT-BOX F = 1.085, P = 0.354
MAXIMUM VARIANCE / MINIMUM VARIANCE = 4.105

ONE-WAY ANOVA ON MAXIMAL OXYGEN CONSUMPTION (POST-TRAINING)

ANALYSIS OF BLOOD SAMPLES
TWO-WAY ANOVA ON BASAL MVO2 %BF EOS & TWBC
FILE NONAME (CREATION DATE = 05/20/79)

05/20/79 PAGE 2

ONEWAY

VARIABLE MVO2

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	80.9993	26.9998	1.410	0.2623
WITHIN GROUPS	25	478.8609	19.1544		
TOTAL	28	559.8601			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	8	63.1000	2.9442	1.0409	56.6000	66.2000	60.6385 TO 65.5614
GRP2	7	64.7142	4.5918	1.7355	58.6000	69.9000	60.4675 TO 68.9610
GRP3	7	64.7428	2.3550	0.8901	62.4000	68.2000	62.5647 TO 66.9208
GRP4	7	60.5714	6.5625	2.4804	53.3000	70.3000	54.5021 TO 66.6406
TOTAL	29	63.2758	4.4716	0.8304	53.3000	70.3000	61.5749 TO 64.9767
	FIXED EFFECTS MODEL		4.3756	0.8127			61.6020 TO 64.9496
	RANDOM EFFECTS MODEL		1.9311	0.9656			60.2030 TO 66.3486

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 1.0834

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.5496, P = 0.045 (APPROX.)
BARTLETT-BOX F = 2.367, P = 0.069
MAXIMUM VARIANCE / MINIMUM VARIANCE = 7.765

ONE -WAY ANOVA ON PERCENT BODY FAT (POST-TRAINING)

ANALYSIS OF BLOOD SAMPLES
TWO-WAY ANOVA ON BASAL MVO2 XBF EOS & TWBC
FILE NONAME (CREATION DATE = 05/20/79)

05/20/79 PAGE 4

- O N E W A Y -

VARIABLE XBF2

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	96.7158	32.2386	2.248	0.1075
WITHIN GROUPS	25	358.5227	14.3409		
TOTAL	28	455.2383			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	8	11.1875	4.3930	1.5532	5.6000	18.7000	7.5149 TO 14.8601
GRP2	7	12.8000	3.1075	1.1745	8.2000	17.0000	9.9260 TO 15.6739
GRP3	7	10.0000	4.5089	1.7042	5.3000	17.2000	5.8300 TO 14.1700
GRP4	7	14.9286	2.6930	1.0179	11.1000	19.5000	12.4380 TO 17.4192
TOTAL	29	12.1931	4.0322	0.7488	5.3000	19.5000	10.6593 TO 13.7269
FIXED EFFECTS MODEL			3.7869	0.7032			10.7448 TO 13.6414
RANDOM EFFECTS MODEL			2.1115	1.0558			8.8333 TO 15.5529

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 2.4716

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.3596, P = 0.547 (APPROX.)
BARTLETT-BOX F = 0.715, P = 0.543
MAXIMUM VARIANCE / MINIMUM VARIANCE = 2.803

T-TEST ON PRE AND POST MAXIMAL SWIM TIMES

ANALYSIS OF BLOOD SAMPLES
T-TEST ON MVO2 ABF MS
FILE NONAME (CREATION DATE = 06/10/79) 06/10/79 PAGE 2

T - T E S T

VARIABLE	NUMBER OF CASES	MEAN	STANDARD DEVIATION	STANDARD ERROR	(DIFFERENCE) MEAN	STANDARD DEVIATION	STANDARD ERROR	CORR.	2-TAIL PROB.	T VALUE	DEGREES OF FREEDOM	2-TAIL PROB.
MS1	29	75.4034	7.566	1.405	7.9379	3.533	0.656	0.893	0.000	12.10	28	0.000
MS2		67.4654	5.828	1.082								

T-TEST ON PRE AND POST MAXIMAL OXYGEN CONSUMPTION AND PERCENT BODY FAT VALUES

ANALYSIS OF BLOOD SAMPLES 06/06/79 PAGE 2
T-TEST ON MVO2 %BF
FILE NONAME (CREATION DATE = 06/06/79)

T - T E S T

VARIABLE	NUMBER OF CASES	MEAN	STANDARD DEVIATION	STANDARD ERROR	(DIFFERENCE) MEAN	STANDARD DEVIATION	STANDARD ERROR	2-TAIL CORR.	2-TAIL PROB.	T VALUE	DEGREES OF FREEDOM	2-TAIL PROB.
MVO1	29	64.6378	5.069	0.941	1.3621	4.331	0.804	0.594	0.001	1.69	28	0.101
MVO2		63.2758	4.471	0.830								
%BF1	29	12.4172	4.126	0.766	0.2241	0.988	0.183	0.971	0.000	1.22	28	0.232
%BF2		12.1931	4.032	0.749								

EOSINOPHIL GROUP MEANS & SD

(PRE-EXERCISE)

		GROUP ONE		GROUP TWO		GROUP THREE		GROUP FOUR	
		$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD
1	0918			0.4050	0.1909	0.2600	0.0141	0.4450	0.2899
2	0920	0.2567	0.1446	0.2683	0.1554	0.2050	0.0622	0.3020	0.2312
3	0922	0.2237	0.1024	0.2343	0.1261	0.1744	0.0606	0.2740	0.2544
4	0925	0.2512	0.1163	0.2286	0.1317	0.1700	0.0394	0.3460	0.2239
5	0927	0.2400	0.1445	0.2657	0.2193	0.2022	0.0740	0.2700	0.1801
6	0929	0.2150	0.1065	0.2143	0.1274	0.1867	0.0742	0.2040	0.1820
7	1002	0.1700	0.0	0.2750	0.1344			0.0933	0.0058
8	1004								
9	1006								
10	1011	0.2725	0.1859	0.2700	0.1299	0.2267	0.0651	0.3375	0.2290
11	1023	0.2100	0.1206	0.2040	0.1184	0.1760	0.0365	0.2325	0.1537
12	1025	0.1680	0.1021	0.1825	0.0465	0.1720	0.0239	0.2450	0.1702
13	1027	0.1480	0.1064	0.1600	0.0374	0.1180	0.0249	0.3250	0.0636
14	1030	0.2000	0.1179	0.1920	0.1295	0.1775	0.0350	0.2350	0.1091
15	1101	0.0925	0.0386	0.1480	0.1156	0.1233	0.0252	0.2475	0.1237
16	1103	0.1275	0.0802	0.2150	0.1159	0.1025	0.0126	0.3500	0.0
17	1106	0.1820	0.1359	0.1800	0.1447	0.1440	0.0616	0.3000	0.0283
18	1108	0.2200	0.1690	0.2025	0.1396	0.3167	0.1258	0.3025	0.1621

EOSINOPHIL GROUP MEANS

(POST-EXERCISE)

	DATE	GROUP ONE		GROUP TWO		GROUP THREE		GROUP FOUR	
		$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD
		CELLS X $10^3/mm^3$							
1	0918*			0.4050	0.1909	0.2600	0.0141	0.4450	0.2899
2	0920	0.2567	0.1446	0.2683	0.1554	0.2050	0.0622	0.3020	0.2312
3	0922	0.2237	0.1024	0.2343	0.1261	0.1744	0.0606	0.2740	0.2544
4	0925	0.2512	0.1163	0.2286	0.1317	0.1700	0.0394	0.3460	0.2239
5	0927	0.2400	0.1445	0.2657	0.2193	0.2022	0.0740	0.2700	0.1801
6	0929	0.2150	0.1065	0.2143	0.1274	0.1867	0.0742	0.2040	0.1820
7	1002**	0.2025	0.0792	0.2043	0.0952	0.1589	0.0788	0.0600	0.0300
8	1004	0.2300	0.1853	0.1750	0.0742	0.1043	0.0489	0.2450	0.2108
9	1006	0.1900	0.1120	0.1529	0.0892	0.1614	0.0682	0.2900	0.2828
10	1011	0.2171	0.1215	0.2050	0.0742	0.1100	0.0583	0.2400	0.1473
11	1023	0.1875	0.1055	0.1586	0.0903	0.1433	0.0216	0.1875	0.1047
12	1025	0.1583	0.1072	0.0983	0.0591	0.1086	0.0567	0.1650	0.1053
13	1027	0.1443	0.0700	0.1300	0.0869	0.0933	0.0602	0.2700	0.1131
14	1030	0.1100	0.0854	0.1300	0.0872	0.0800	0.0443	0.1267	0.0808
15	1101	0.0967	0.0592	0.1071	0.0743	0.0633	0.0115	0.1585	0.1672
16	1103	0.1229	0.0765	0.1280	0.0722	0.0800	0.0438	0.2800	0.0707
17	1106	0.1550	0.0787	0.1143	0.0707	0.1060	0.0669	0.2200	0.0424
18	1108	0.1929	0.1661	0.1633	0.1031	0.1600	0.0872	0.2150	0.1179

* BASAL SAMPLES

** TRAINING SAMPLES

TOTAL WHITE BLOOD CELL

GROUP MEANS & STANDARD DEVIATION

(PRE-EXERCISE)

		GROUP ONE		GROUP TWO		GROUP THREE		GROUP FOUR	
		$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD
1	0918	6.6813		7.2850	0.9546	6.7650	1.3789	8.2200	1.0889
2	0920	6.6783	1.1134	6.8450	0.7749	6.7883	1.8251	6.0360	1.0773
3	0922	5.8350	1.0363	6.3757	0.8791	6.1267	1.4255	5.5360	1.3184
4	0925	5.8575	0.9108	5.5171	0.5650	6.0078	1.6365	5.8200	1.2097
5	0927	6.0214	1.4435	6.4100	0.9534	6.1956	1.8308	6.2300	1.1637
6	0929	5.8825	1.1945	6.2000	1.1731	5.8678	2.0455	5.8380	1.5948
7	1002	3.8700	0.0	5.2550	0.4879			4.6600	0.3659
8	1004								
9	1006								
10	1011	6.6225	0.8934	6.3600	1.3144	7.0333	3.2316	6.4025	1.4152
11	1023	6.1700	0.5961	6.6000	0.8387	6.5460	1.8074	6.1375	1.9041
12	1025	6.0760	0.9767	6.1860	1.2757	6.3080	1.4436	5.3100	0.8977
13	1027	5.7540	0.9507	6.3825	0.4233	7.4180	4.4416	7.1850	0.2758
14	1030	6.2560	1.0391	6.5680	1.0773	7.4600	2.2514	8.2775	0.8574
15	1101	4.9125	0.7346	4.9120	0.7462	5.2733	1.3151	6.0250	0.8095
16	1103	4.1950	0.6582	5.3100	1.3730	4.8900	1.1016	6.9100	0.0
17	1106	5.3440	0.9565	4.9340	0.9864	5.2150	1.0411	6.3100	1.9375
18	1108	6.8240	0.5412	7.8750	0.9782	8.9833	2.2007	7.0325	1.3724

TOTAL WHITE BLOOD CELL

MEANS & STANDARD DEVIATION

PER SAMPLE DAY

(POST-EXERCISE)

		GROUP ONE		GROUP TWO		GROUP THREE		GROUP FOUR	
		$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD	$\bar{X}$	± SD
1	0918*	6.6813		7.2850	0.9546	6.7650	1.3789	8.2200	1.0889
2	0920	6.6783	1.1134	6.8450	0.7749	6.7883	1.8251	6.0360	1.0773
3	0922	5.8350	1.0363	6.3757	0.8791	6.1267	1.4255	5.5360	1.3184
4	0925	5.8575	0.9108	5.5171	0.5650	6.0078	1.6365	5.8200	1.2097
5	0927	6.0214	1.4435	6.4100	0.9534	6.1956	1.8308	6.2300	1.1637
6	0929	5.8825	1.1945	6.2000	1.1731	5.8678	2.0455	5.8380	1.5948
7	1002**	6.4462	2.6117	5.6286	1.3517	6.4611	2.0438	4.5200	0.5786
8	1004	7.7587	1.8640	7.9529	2.1747	7.3071	1.1266	6.2975	3.0250
9	1006	6.7025	1.5770	5.5417	2.3151	7.0114	1.5030	7.8700	3.5780
10	1011	7.3814	2.6705	6.3200	1.0560	6.7940	1.9770	7.7200	3.1717
11	1023	7.1562	0.9693	7.5443	1.7612	6.8817	1.9356	6.5125	1.3075
12	1025	7.4567	2.1613	6.9283	1.5354	6.4357	1.7773	5.3975	1.3732
13	1027	5.7100	1.6652	5.9660	1.8949	5.9717	2.8528	6.5100	1.5839
14	1030	6.4800	0.9923	7.2567	1.1719	6.5600	2.2656	8.3400	1.2150
15	1101	4.7433	1.1817	5.1943	0.8853	4.9067	1.6974	6.0575	1.7256
16	1103	5.2114	1.4976	5.3260	0.6540	4.9950	1.0573	7.4100	2.4607
17	1106	6.7833	1.7880	7.6000	1.6723	5.6820	1.9442	6.8650	2.6658
18	1108	8.4857	2.5838	8.6450	0.6983	6.9075	2.0846	8.1700	2.4227

* BASAL SAMPLES

** TRAINING SAMPLES

TWO-WAY ANOVA ON PRE -EXERCISE EOSINOPHIL LEVELS

ANALYSIS OF BLOOD SAMPLES 05/08/79 PAGE 2
TWO-WAY ANOVA ON TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/08/79)

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

EOS
BY INTENS
LOAD

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	0.139	2	0.069	5.543	0.005
INTENS	0.034	1	0.034	2.713	0.102
LOAD	0.110	1	0.110	8.759	0.004
2-WAY INTERACTIONS	0.084	1	0.084	6.715	0.011
INTENS LOAD	0.084	1	0.084	6.715	0.011
EXPLAINED	0.223	3	0.074	5.934	0.001
RESIDUAL	1.790	143	0.013		
TOTAL	2.012	146	0.014		

148 CASES WERE PROCESSED.
1 CASES (0.7 PCT) WERE MISSING.

TWO-WAY ANOVA ON PRE -EXERCISE TOTAL WHITE BLOOD CELL LEVELS

ANALYSIS OF BLOOD SAMPLES
TWO-WAY ANOVA ON TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/08/79)

05/08/79 PAGE 4

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

TWBC
BY INTENS
LOAD

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	15.220	2	7.610	2.949	0.056
INTENS	14.076	1	14.076	5.455	0.021
LOAD	1.498	1	1.498	0.581	0.447
2-WAY INTERACTIONS	0.290	1	0.290	0.112	0.738
INTENS LOAD	0.290	1	0.290	0.112	0.738
EXPLAINED	15.510	3	5.170	2.003	0.116
RESIDUAL	369.022	143	2.581		
TOTAL	384.531	146	2.634		

148 CASES WERE PROCESSED.
1 CASES (0.7 PCT) WERE MISSING.

TWO-WAY ANOVA ON POST-EXERCISE EOSINOPHIL LEVELS

ANALYSIS OF BLOCD SAMPLES
TWO-WAY ANOVA ON TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/10/79)

05/10/79 PAGE 2

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

EOS
BY GRP
TEST

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	0.632	4	0.158	14.903	0.000
GRP	0.433	3	0.144	13.622	0.000
TEST	0.167	1	0.167	15.714	0.000
2-WAY INTERACTIONS	0.040	3	0.013	1.246	0.293
GRP TEST	0.040	3	0.013	1.246	0.293
EXPLAINED	0.671	7	0.096	9.050	0.000
RESIDUAL	3.964	374	0.011		
TOTAL	4.635	391	0.012		

384 CASES WERE PROCESSED.
2 CASES (0.5 PCT) WERE MISSING.

TWO-WAY ANOVA ON POST-EXERCISE TOTAL WHITE BLOOD CELL LEVELS

ANALYSIS OF BLOOD SAMPLES
TWO-WAY ANOVA ON TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/10/79) 05/10/79 PAGE 4

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

TWBC
BY GRP
TEST

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	26.701	4	6.675	1.983	0.096
GRP	8.322	3	2.774	0.824	0.481
TEST	19.921	1	19.921	5.919	0.015
2-WAY INTERACTIONS	15.586	3	5.195	1.544	0.203
GRP TEST	15.586	3	5.195	1.544	0.203
EXPLAINED	42.288	7	6.041	1.795	0.087
RESIDUAL	1258.769	374	3.366		
TOTAL	1301.057	381	3.415		

384 CASES WERE PROCESSED.
2 CASES (0.5 PCT) WERE MISSING.

ONE-WAY ANOVA ON EOSINOPHIL LEVELS COMPARING PRE AND POST EXERCISE VALUES

- O N E W A Y -

VARIABLE EOS

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	1	0.1986	0.1986	17.010	0.0000
WITHIN GROUPS	380	4.4363	0.0117		
TOTAL	381	4.6349			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	147	0.1997	0.1174	0.0097	0.0100	0.5800	0.1806 TO 0.2189
GRP2	235	0.1529	0.1018	0.0066	0.0100	0.5700	0.1398 TO 0.1659
TOTAL	382	0.1709	0.1103	0.0056	0.0100	0.5800	0.1598 TO 0.1820
FIXED EFFECTS MODEL		0.1080	0.0055				0.1600 TO 0.1818
RANDOM EFFECTS MODEL		0.0339	0.0240				-0.1337 TO 0.4755

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 0.0010

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.5709, P = 0.025 (APPROX.)
BARTLETT-BOX F = 3.726, P = 0.054
MAXIMUM VARIANCE / MINIMUM VARIANCE = 1.331

ONE-WAY ANOVA ON TOTAL WHITE BLOOD CELL

LEVELS COMPARING PRE AND POST EXERCISE VALUES

- O N E W A Y -

VARIABLE TWBC

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	1	19.0539	19.0539	5.663	0.0178
WITHIN GROUPS	382	1284.0977	3.3613		
TOTAL	383	1303.1514			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	148	6.2356	1.6207	0.1332	3.4200	15.2000	5.9724 TO 6.4988
GRP2	236	6.6933	1.9550	0.1273	0.8520	12.4000	6.4425 TO 6.9440
TOTAL	384	6.5169	1.8886	0.0941	0.8520	15.2000	6.3319 TO 6.7019
	FIXED EFFECTS MODEL		1.8334	0.0936			6.3329 TO 6.7008
	RANDOM EFFECTS MODEL		0.3291	0.2327			3.5601 TO 9.4736

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 0.0363

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.5928, P = 0.000 (APPROX.)
BARTLETT-BOX F = 6.153, P = 0.013
MAXIMUM VARIANCE / MINIMUM VARIANCE = 1.456

THREE-WAY ANOVA ON EOSINOPHIL LEVELS

FILE NONAME (CREATION DATE = 05/10/79)

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

EOS
BY INTENS
LOAD
TEST

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	0.320	3	0.107	10.075	0.000
INTENS	0.006	1	0.006	0.570	0.451
LOAD	0.119	1	0.119	11.247	0.001
TEST	0.183	1	0.183	17.283	0.000
2-WAY INTERACTIONS	0.347	3	0.116	10.908	0.000
INTENS LOAD	0.295	1	0.295	27.871	0.000
INTENS TEST	0.021	1	0.021	1.994	0.159
LOAD TEST	0.016	1	0.016	1.542	0.215
3-WAY INTERACTIONS	0.005	1	0.005	0.446	0.505
INTENS LOAD TEST	0.005	1	0.005	0.446	0.505
EXPLAINED	0.672	7	0.096	9.056	0.000
RESIDUAL	3.963	374	0.011		
TOTAL	4.635	381	0.012		

380 CASES WERE PROCESSED.
2 CASES (0.5 PCT) WERE MISSING.

THREE-WAY ANOVA ON TOTAL WHITE BLOOD CELL LEVELS

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

TWBC
BY INTENS
LOAD
TEST

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	24.751	3	8.250	2.451	0.063
INTENS	2.120	1	2.120	0.630	0.428
LOAD	4.734	1	4.734	1.407	0.236
TEST	19.509	1	19.509	5.797	0.017
2-WAY INTERACTIONS	14.791	3	4.930	1.465	0.224
INTENS LOAD	1.458	1	1.458	0.433	0.511
INTENS TEST	12.667	1	12.667	3.764	0.053
LOAD TEST	0.017	1	0.017	0.005	0.944
3-WAY INTERACTIONS	2.808	1	2.808	0.834	0.362
INTENS LOAD TEST	2.808	1	2.808	0.834	0.362
EXPLAINED	42.351	7	6.050	1.798	0.086
RESIDUAL	1258.706	374	3.366		
TOTAL	1301.057	381	3.415		

384 CASES WERE PROCESSED.
2 CASES (0.5 PCT) WERE MISSING.

ANALYSIS OF BLOOD SAMPLES
A ONE-WAY ANCVA ON ALL TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/08/79)

- O N E W A Y -

VARIABLE EOS

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	0.2228	0.0743	5.934	0.0003
WITHIN GROUPS	143	1.7896	0.0125		
TOTAL	146	2.0124			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	42	0.1812	0.1222	0.0189	0.0400	0.5300	0.1431 TO 0.2193
GRP2	40	0.1935	0.1089	0.0172	0.0100	0.4300	0.1587 TO 0.2283
GRP3	36	0.1669	0.0723	0.0121	0.0800	0.4500	0.1425 TO 0.1914
GRP4	29	0.2759	0.1377	0.0256	0.0800	0.5800	0.2235 TO 0.3282
TOTAL	147	0.1997	0.1174	0.0097	0.0100	0.5800	0.1806 TO 0.2189
FIXED EFFECTS MODEL			0.1119	0.0092			0.1815 TO 0.2180
RANDOM EFFECTS MODEL			0.0454	0.0227			0.1275 TO 0.2720

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 0.0017

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.3719, P = 0.038 (APPROX.)
BARTLETT-BOX F = 4.508, P = 0.004
MAXIMUM VARIANCE / MINIMUM VARIANCE = 3.625

THE SCHEFFE MULTIPLE RANGE TEST ON EOSINOPHIL LEVELS

ANALYSIS OF BLOOD SAMPLES 05/08/79 PAGE 3
A ONE-WAY ANCVA ON ALL TRAINING EOS & TEBC
FILE NONAME (CREATION DATE = 05/08/79)

- O N E W A Y -

VARIABLE EOS

MULTIPLE RANGE TEST

SCHEFFE PROCEDURE
RANGES FOR THE 0.050 LEVEL -

4.00 4.00 4.00

THE RANGES ABOVE ARE TABLE RANGES. THE VALUE ACTUALLY COMPARED WITH MEAN(J)-MEAN(I) IS..
0.0791 * RANGE * SQRT(1/N(I) + 1/N(J))

HOMOGENEOUS SUBSETS (SUBSETS OF GROUPS, WHOSE HIGHEST AND LOWEST MEANS DO NOT DIFFER BY MORE THAN THE SHORTEST SIGNIFICANT RANGE FOR A SUBSET OF THAT SIZE)

SUBSET 1

GROUP	GRP3	GRP1	GRP2
MEAN	0.1669	0.1812	0.1935

SUBSET 2

GROUP	GRP4
MEAN	0.2759

ONE -WAY ANOVA ON PRE-EXERCISE TOTAL WHITE BLOOD CELL LEVELS

ANALYSIS OF BLOOD SAMPLES 05/08/79 PAGE 7
A ONE-WAY ANOVA ON ALL TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/08/79)

- O N E W A Y -

VARIABLE TWBC

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	15.8008	5.2669	2.049	0.1096
WITHIN GROUPS	144	370.0846	2.5700		
TOTAL	147	385.8853			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	42	5.8343	1.0800	0.1667	3.5500	7.7200	5.4977 TO 6.1708
GRP2	41	6.0905	1.2870	0.2010	3.4200	9.2900	5.6843 TO 6.4967
GRP3	36	6.5414	2.4221	0.4037	4.1900	15.2000	5.7219 TO 7.3609
GRP4	29	6.6428	1.3454	0.2498	4.1800	8.9100	6.1310 TO 7.1545
TOTAL	148	6.2357	1.6202	0.1332	3.4200	15.2000	5.9725 TO 6.4989
	FIXED EFFECTS MODEL		1.6031	0.1318			5.9752 TO 6.4961
	RANDOM EFFECTS MODEL		0.3798	0.1899			5.6313 TO 6.8400

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 0.0734

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.5587, P = 0.000 (APPROX.)
BARTLETT-BOX F = 10.183, P = 0.000
MAXIMUM VARIANCE / MINIMUM VARIANCE = 5.029

THE SCHEFFE MULTIPLE RANGE TEST ON TOTAL WHITE BLOOD CELL LEVELS

ANALYSIS OF BLOOD SAMPLES
A ONE-WAY ANOVA ON ALL TRAINING EGS A TWBC
FILE NONAME (CREATION DATE = 05/08/79)

- O N E W A Y -

VARIABLE TWBC

MULTIPLE RANGE TEST

SCHEFFE PROCEDURE
RANGES FOR THE C.050 LEVEL -

4.00 4.00 4.00

THE RANGES ABOVE ARE TABLE RANGES. THE VALUE ACTUALLY COMPARED WITH MEAN(J)-MEAN(I) IS..
1.1336 * RANGE * SQRT(1/N(I) + 1/N(J))

HOMOGENEOUS SUBSETS (SUBSETS OF GROUPS, WHOSE HIGHEST AND LOWEST MEANS DO NOT DIFFER BY MORE THAN THE SHORTEST SIGNIFICANT RANGE FOR A SUBSET OF THAT SIZE)

SUBSET 1

GROUP	GRP1	GRP2	GRP3	GRP4
MEAN	5.8343	6.0905	6.5414	6.6428

TWO-WAY ANOVA ON EOSINOPHIL LEVELS (POST-EXERCISE)

ANALYSIS OF BLOOD SAMPLES
TWO-WAY ANOVA ON TRAINING EOS & TABC
FILE NONAME (CREATION DATE = 05/08/79)

05/08/79 PAGE 2

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

EOS
BY INTENS
LOAD

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	0.034	2	0.017	1.812	0.166
INTENS	0.003	1	0.003	0.314	0.576
LOAD	0.029	1	0.029	3.051	0.082
2-WAY INTERACTIONS	0.216	1	0.216	22.955	0.000
INTENS LOAD	0.216	1	0.216	22.955	0.000
EXPLAINED	0.250	3	0.083	8.859	0.000
RESIDUAL	2.174	231	0.009		
TOTAL	2.424	234	0.010		

236 CASES WERE PROCESSED.
1 CASES (0.4 PCT) WERE MISSING.

TWO-WAY ANOVA ON TOTAL WHITE BLOOD CELL LEVELS (POST-EXERCISE)

* * * * * * * * * * A N A L Y S I S O F V A R I A N C E * * * * * * * * * *

TWBC
BY INTENS
LOAD

* *

SOURCE OF VARIATION	SUM OF SQUARES	DF	MEAN SQUARE	F	SIGNIF OF F
MAIN EFFECTS	4.460	2	2.230	0.579	0.561
INTENS	1.277	1	1.277	0.332	0.565
LOAD	2.720	1	2.720	0.706	0.402
2-WAY INTERACTIONS	3.987	1	3.987	1.035	0.310
INTENS LOAD	3.987	1	3.987	1.035	0.310
EXPLAINED	8.447	3	2.816	0.731	0.534
RESIDUAL	889.682	231	3.851		
TOTAL	898.129	234	3.838		

236 CASES WERE PROCESSED.
1 CASES (0.4 PCT) WERE MISSING.

ONE-WAY ANOVA ON POST-EXERCISE EOSINOPHIL LEVELS

ANALYSIS OF BLOOD SAMPLES (BY GROUP) 05/08/79 PAGE 2
A ONE-WAY ANOVA ON ALL TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/08/79)

- O N E W A Y -

VARIABLE EOS

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	0.2500	0.0833	8.854	0.0000
WITHIN GROUPS	231	2.1740	0.0094		
TOTAL	234	2.4240			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	73	0.1697	0.1157	0.0135	0.0200	0.5700	0.1427 TO 0.1967
GRP2	65	0.1425	0.0805	0.0100	0.0100	0.3100	0.1225 TO 0.1624
GRP3	62	0.1115	0.0587	0.0075	0.0300	0.2500	0.0965 TO 0.1264
GRP4	35	0.2104	0.1311	0.0222	0.0340	0.4900	0.1654 TO 0.2554
TOTAL	235	0.1529	0.1013	0.0066	0.0100	0.5700	0.1398 TO 0.1659
FIXED EFFECTS MODEL			0.0970	0.0063			0.1404 TO 0.1653
RANDOM EFFECTS MODEL			0.0990	0.0195			0.0908 TO 0.2149

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE 0.0013

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.4242, P = 0.000 (APPROX.)
BARTLETT-BOX F = 12.971, P = 0.000
MAXIMUM VARIANCE / MINIMUM VARIANCE = 4.963

THE SCHEFFE MULTIPLE RANGE TEST ON EOSINOPHIL LEVELS

ANALYSIS OF BLOOD SAMPLES 05/08/79 PAGE 3
A ONE-WAY ANOVA ON ALL TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/08/79)

- - - -

ONE-WAY ANOVA ON POST-EXERCISE TOTAL WHITE BLOOD CELL LEVELS

ANALYSIS OF BLOOD SAMPLES (BY GROUP) 05/08/79 PAGE 7
A ONE-WAY ANOVA ON ALL TRAINING EOS & TWBC
FILE NONAME (CREATION DATE = 05/08/79)

- O N E W A Y -

VARIABLE TWBC

ANALYSIS OF VARIANCE

SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARES	F RATIO	F PROB.
BETWEEN GROUPS	3	8.3882	2.7961	0.729	0.5356
WITHIN GROUPS	232	889.8443	3.8355		
TOTAL	235	898.2324			

GROUP	COUNT	MEAN	STANDARD DEVIATION	STANDARD ERROR	MINIMUM	MAXIMUM	95 PCT CONF INT FOR MEAN
GRP1	73	6.7643	2.0221	0.2367	3.2000	12.4000	6.2925 TO 7.2361
GRP2	66	6.7639	1.8515	0.2279	0.8520	12.0000	6.3088 TO 7.2191
GRP3	62	6.3945	1.8743	0.2380	3.7800	11.7000	5.9185 TO 6.8705
GRP4	35	6.9414	2.1579	0.3647	3.9000	11.7000	6.2002 TO 7.6827
TOTAL	236	6.6933	1.9551	0.1273	0.8520	12.4000	6.4426 TO 6.9440
FIXED EFFECTS MODEL			1.9585	0.1275			6.4421 TO 6.9445
RANDOM EFFECTS MODEL			0.2550	0.1275			6.2876 TO 7.0990

WARNING - BETWEEN COMPONENT VARIANCE IS NEGATIVE
IT WAS REPLACED BY 0.0 IN COMPUTING ABOVE RANDOM EFFECTS MEASURES

RANDOM EFFECTS MODEL - ESTIMATE OF BETWEEN COMPONENT VARIANCE -0.0180

TESTS FOR HOMOGENEITY OF VARIANCES

COCHRANS C = MAX. VARIANCE/SUM(VARIANCES) = 0.2968, P = 0.372 (APPROX.)
BARTLETT-BOX F = 0.482, P = 0.695
MAXIMUM VARIANCE / MINIMUM VARIANCE = 1.358

APPENDIX 'C'

ADDITIONAL INFORMATION ON THE HEMALOG-D ANALYZER

6-1 INTRODUCTION

This Section provides both a general and a detailed description of the Technicon Hemalog D automated leukocyte differential analyzer.

6-2 GENERAL DESCRIPTION

The Hemalog D system (Figure 6-1) is an automated differential white blood cell counter that performs 10 000 cell differential counts on 0.369-ml samples of EDTA anticoagulated venous blood at the rate of 60 samples per hour

The Technicon Hemalog D system consists of three channels; the peroxidase channel, the monocyte channel, and the basophil channel. Each channel contains its own manifold, flowcell, and electro-optical system.

While each channel utilizes a different cytochemical reaction, characteristic of the type(s) of leukocytes to be determined, the operation of each channel can be summarized in four basic procedures:

Fixing – Since many cell enzymes are easily denatured or altered, each channel uses a fixing agent to immobilize the cellular enzymes quickly and uniformly without significantly affecting their enzymatic activity.

Lysing – In normal blood, red cells vastly outnumber white cells. Since one or more red cells passing through the detection system might be mistaken for a leukocyte, each channel contains a red cell lysing agent.

Staining – The staining step is performed to characteristically "tag" a leukocyte for detection in the electro-optical system. Reaction conditions such as temperature and pH are closely controlled to ensure maximum specificity. The preferential stains used in the Technicon Hemalog D system are summarized in Table 6-1.

Detection – After staining, each sample passes through a sheath-stream flowcell where photodetectors measure the absorbance properties and the forward-scattering signature of each cell type. The electrical signals generated by the photodectors are processed by logic circuits that determine the cell type and quantity.

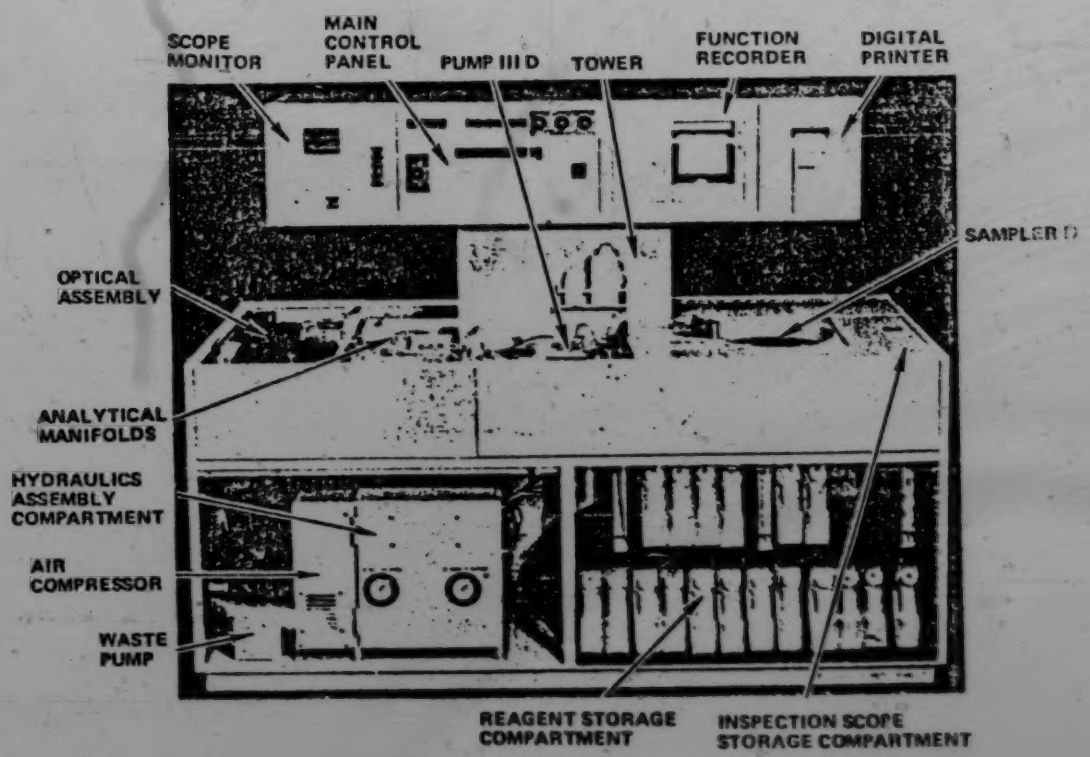


Figure 6-1 TECHNICON HEMALOG D SYSTEM

Table 6-1 SUMMARY OF TECHNICON HEMALOG D STAINING METHODS

CELL TYPE	STAINING METHOD (Refer to notes 1, 2, 3, and 4.)		
	PEROXIDASE	ESTERASE	ALCIAN BLUE
Neutrophils	+	+	–
Eosinophils	++	–	–
Basophils	–	–	+
Monocytes	±	+,++	–
Lymphocytes	–	–	–

NOTE

1. "+" indicates a detectable staining of the listed cell type.
2. "++" indicates a deep staining of the listed cell type.
3. "–" indicates no detectable staining of the listed cell types.
4. "±" indicates very light and inconsistent staining of the listed cell type.

The results for each specimen are provided on a preprinted report form (Figure 6-2). Each differential white blood cell count is reported in terms of its absolute number per cubic millimeter of sample and in terms of its percentage of the total leukocyte population. The total WBC count per cubic millimeter of sample also is provided on this report form. Samples with total WBC counts greater than 20 000 cells per cubic millimeter (beyond the range of this instrument) are identified by a repeating "HI" symbol. Note that on the original copy of the preprinted report form, the High Peroxidase Activity (HPX) is provided. The HPX value, the low rate (LR) indication, and the Low Peroxidase indication are described fully in this Section. In addition to the analytical data, the date, sample sequence number, the the Technicon IDee sample identification number (optional) are printed on the preprinted report form.

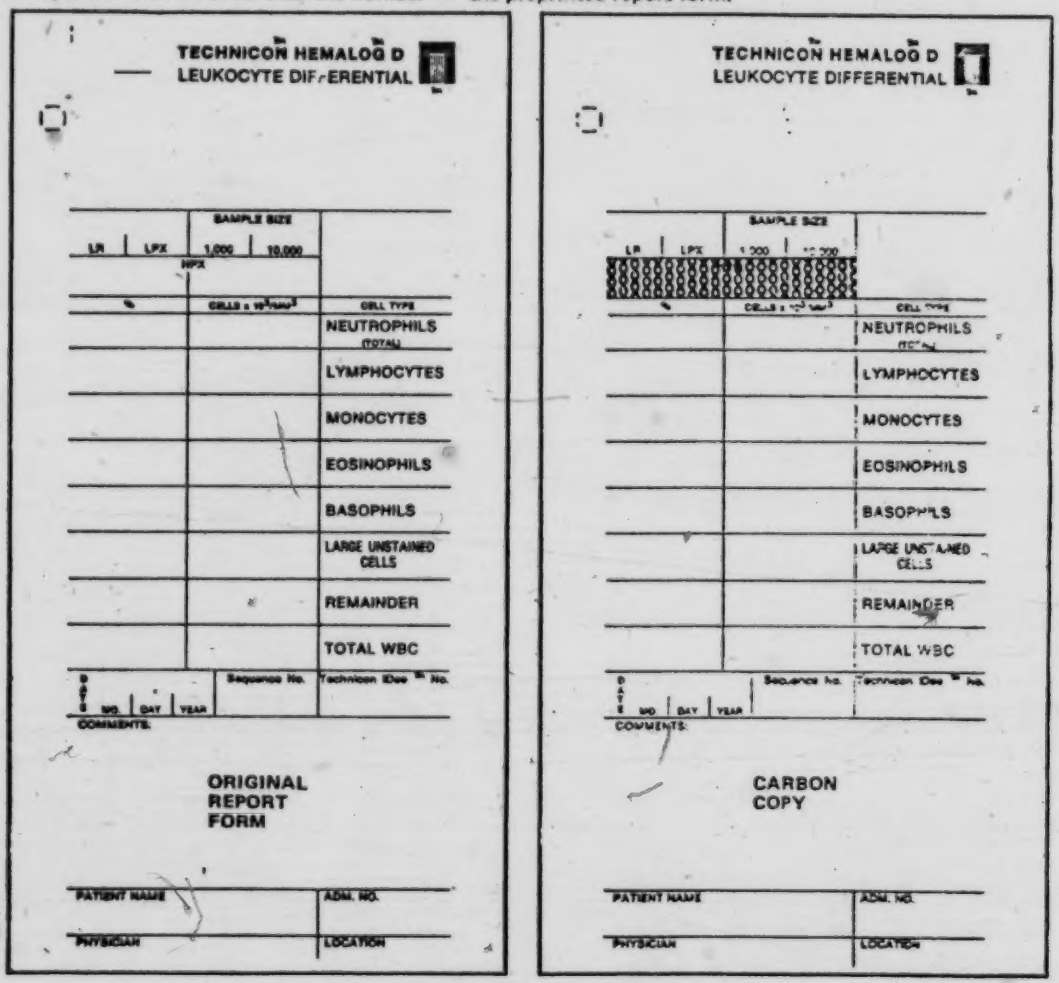


Figure 6-2 PREPRINTED REPORT FORM

Section VII
SAMPLE COLLECTION AND PREPARATION

TABLE OF CONTENTS

Title	Page
Patient Preparation	7-1
Sample Collection	7-1
Sample Preparation	7-1
Sample Storage	7-1
Sample Stability	7-1

TABLE

Table		Page
7-1	Leukocyte Stability at Room Temperature	7-1

7-1 PATIENT PREPARATION

Patients should be prepared according to the protocol established by the user's laboratory.

7-2 SAMPLE COLLECTION

The whole blood sample should be collected using routine venipuncture techniques. It is recommended that the sample be collected in a 5-, 7-, or 10-ml Vacutainer* (lavender tops) containing EDTA (K_3) as the anticoagulant.

7-3 SAMPLE PREPARATION

Other than being collected in the specified collection tube, no special treatment of the whole blood specimen is required.

7-4 SAMPLE STORAGE

It is recommended that the whole blood specimens be stored at room temperature. DO NOT USE COLD (REFRIGERATED) SPECIMENS! If the specimen has been stored in a refrigerator, it must be allowed to equilibrate slowly to room temperature before stirring.

IMPORTANT

If the whole blood specimen is brought to room temperature quickly, there is a strong tendency for the platelets to clump upon stirring.

DO NOT USE PREVIOUSLY FROZEN WHOLE BLOOD AS SAMPLES. Freezing the blood can disrupt cellular structure; thus resulting in aberrant differential counts.

7-5 SAMPLE STABILITY

Studies conducted by Technicon indicated that all morphologically identifiable leukocyte cell types detected by the Technicon™ Hemalog™ D automated leukocyte differential analyzer are stable for 24 hours. The specific stability for each leukocyte result reported by the system is provided in Table 7-1.

Table 7-1 LEUKOCYTE STABILITY AT ROOM TEMPERATURE

RESULT	STABILITY (HOURS)
Neutrophils	48
Eosinophils	72
Lymphocytes	48
Total White Blood Cells	24
Large Unstained Cells	24
High Peroxidase Activity	8
Basophils	72
Monocytes	24
Remainder	48

* Trademark of Becton, Dickinson and Co., Rutherford, NJ

Table 10-1 RANGES OF EXPECTED VALUES

CELL TYPE	STATISTICAL DISTRIBUTION			
	2.5 PERCENTILE	50 PERCENTILE	97.5 PERCENTILE	
Neutrophils (%)	47.3	60.5	75.0	Institution 1*
	44.0	56.8	72.3	Institution 2*
	39.8	58.5	76.6	Institution 3*
Eosinophils (%)	0.5	1.9	5.8	Institution 1
	0.4	1.9	6.6	Institution 2
	0.5	2.5	6.7	Institution 3
Lymphocytes (%)	17.6	30.8	44.5	Institution 1
	18.6	32.7	48.9	Institution 2
	14.9	31.6	47.8	Institution 3
Basophils (%)	0.1	0.6	1.4	Institution 1
	0.3	0.8	1.8	Institution 2
	0.2	0.7	1.3	Institution 3
Monocytes (%)	2.0	6.5	10.9	Institution 1
	0.5	6.2	11.3	Institution 2
	0.2	5.8	11.1	Institution 3
Total WBC ($x10^3$ cells/mm^3)	4.2	6.8	11.3	Institution 1
	3.7	6.1	10.0	Institution 2
	3.8	6.4	12.0	Institution 3
Large Unstained Cells (%)	0.15	1.0	2.55	Institution 1
	0.70	1.6	3.35	Institution 2
	0.65	1.25	3.75	Institution 3
High Peroxidase Activity	0.2	0.4	1.55	Institution 1
	0.1	0.2	0.74	Institution 2
	0.25	0.55	2.5	Institution 3
Remainder (%)	-5.55	0.3	6.05	Institution 1
	-7.20	-1.7	3.60	Institution 2
	-6.15	-1.4	2.65	Institution 3

*The data for the ranges of expected values were obtained from evaluation sites located at a large university referral hospital, at a medical school, and at a medical school with a large outpatient referral center. The names of these institutions are available upon request.

10-2 CALCULATION OF RESULTS

Technicon™ Hemalog™ D automated leukocyte differential analyzers automatically perform all calculations necessary for obtaining final results. All Technicon Hemalog D systems print out the differential cell counts on a preprinted report form in terms of a percentage of the total leukocyte population assayed (10 000 leukocytes or 1 000 leukocytes) and in terms of cells per cubic millimeter of specimen. Additionally, the total number of white blood cells per cubic millimeter is indicated on the preprinted report form.

It is recommended that the technologist observe the monocyte and remainder results for each sample. If the sum

Table 11-2 INDEX OF PRECISION SCATTERGRAMS

Figure	TECHNIQUE	TEST
11-1	Hemalog D system	Neutrophils
11-2	100-Cell Eye Count	Neutrophils
11-3	Hemalog D system	Lymphocytes
11-4	100-Cell Eye Count	Lymphocytes
11-5	Hemalog D system	Monocytes
11-6	100-Cell Eye Count	Monocytes
11-7	Hemalog D system	Eosinophils
11-8	100-Cell Eye Count	Eosinophils
11-9	Hemalog D system	Basophils
11-10	100-Cell Eye Count	Basophils
11-11	Hemalog D system	Total WBC
11-12	Hemalog D system	Large Unstained Cells (LUC)
11-13	Hemalog D system	High Peroxidase Activity (HPX)

11-2 ACCURACY

Introduction

The conventional means for measuring accuracy, the comparison of any method to a reference method, does not apply directly to Technicon Hemalog D system. The classic reference method, a differential white blood cell count done microscopically by eye on a Wright-stained blood film, has several systematic errors. These include the cell distribution errors on the slides and recognition errors with respect to cell types on the slide.

It is not an easy task to isolate the effect of poor precision from systematic errors. In the following figures, this attempt is made by use of simulated ideal distributions that include only those errors due to imprecision. These simulations were made by superimposing the random standard deviations from 100-cell, 200-cell, or 10 000-cell counts on idealized, but realistic, cell distributions. If the clinical data contain only these statistical errors, the clinical and simulated data should have very similar scattergrams. Any significant difference between simulated and clinical data therefore needs interpretation as a deviation from the ideal accuracy of either method. Table 11-3 provides an index of the figures that are used to depict the accuracy of each channel.

Table 11-3 INDEX OF ACCURACY SCATTERGRAMS

Figure	COMPARISON	TEST	TYPE OF POPULATION
11-15	Hemalog D system vs 200-Cell Eye Count	Neut	Clinical
11-16	Hemalog D system vs 100-Cell Eye Count	Neut	Simulated
11-17	Hemalog D system vs 200-Cell Eye Count	Neut	Simulated
11-18	Hemalog D system vs 200-Cell Eye Count	Lymph	Clinical
11-19	Hemalog D system vs 100-Cell Eye Count	Lymph	Simulated
11-20	Hemalog D system vs 200-Cell Eye Count	Lymph	Simulated
11-21	Hemalog D system vs 200-Cell Eye Count	Mono	Clinical
11-22	Hemalog D system vs 100-Cell Eye Count	Mono	Simulated
11-23	Hemalog D system vs 200-Cell Eye Count	Mono	Simulated
11-24	Hemalog D system vs 200-Cell Eye Count	Eos	Clinical
11-25	Hemalog D system vs 100-Cell Eye Count	Eos	Simulated
11-26	Hemalog D system vs 200-Cell Eye Count	Eos	Simulated
11-27	Hemalog D system vs 200-Cell Eye Count	Baso	Clinical
11-28	Hemalog D system vs 100-Cell Eye Count	Baso	Simulated
11-29	Hemalog D system vs 200-Cell Eye Count	Baso	Simulated
11-30	Hemalog D system vs Electronic Cell Counter	Total WBC	Simulated
11-31	Hemalog D system vs Electronic Cell Counter	Total WBC	Clinical

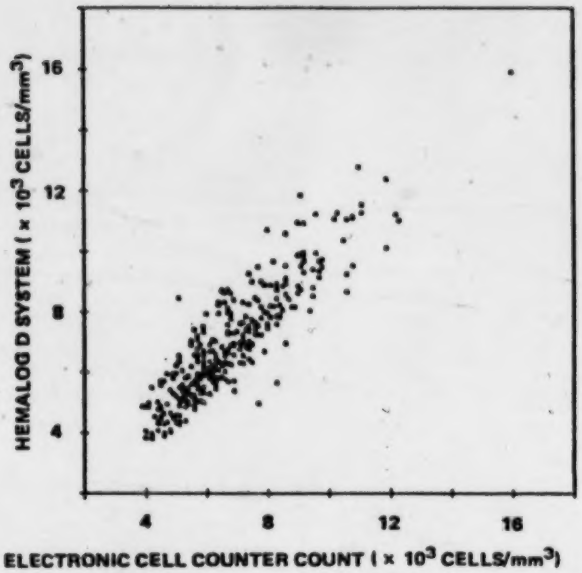


Figure 11-31
TOTAL WBC COUNT COUNT CORRELATION – TECHNICON HEMALOG D SYSTEM VS ELECTRONIC CELL COUNTER COUNT

11-4 SAMPLE INTERACTION

The sample interaction (carryover) of the Total WBC channel was measured by estimating the portion of a given specimen that was lost to the following specimen. This was done by fitting the data to a complex expression and computing the percentage of carryover by multiple-regression analysis.* The study indicated that the amount of carryover measured (1.3%) was not clinically significant in samples with comparable cell counts.

11-5 DRIFT

The amount of drift in test results that could occur over a four-hour period is illustrated in Figure 11-32. The data indicated that there is no significant drift for any channel except LUC. This drift may be caused by the loss of cells due to the aging of the blood during the four hours of the test.

*The technique of multiple-regression analysis is soon to be published. A copy of this paper may be obtained upon request, from Technicon Instruments Corporation, Tarrytown, New York.

11-6 SENSITIVITY OF RESPONSE

The dilution data for the Total WBC, neutrophils, and lymphocytes channels are illustrated in Figures 11-33, 11-34, and 11-35. While the data presented by these Figures appear to be offset from the ideal line, this bias appears to be caused by an original dilution error. The data indicate that Technicon Hemalog D system is an efficient counter of cells with a linear response (based on 10 000 cells) to as few as 500 cells.

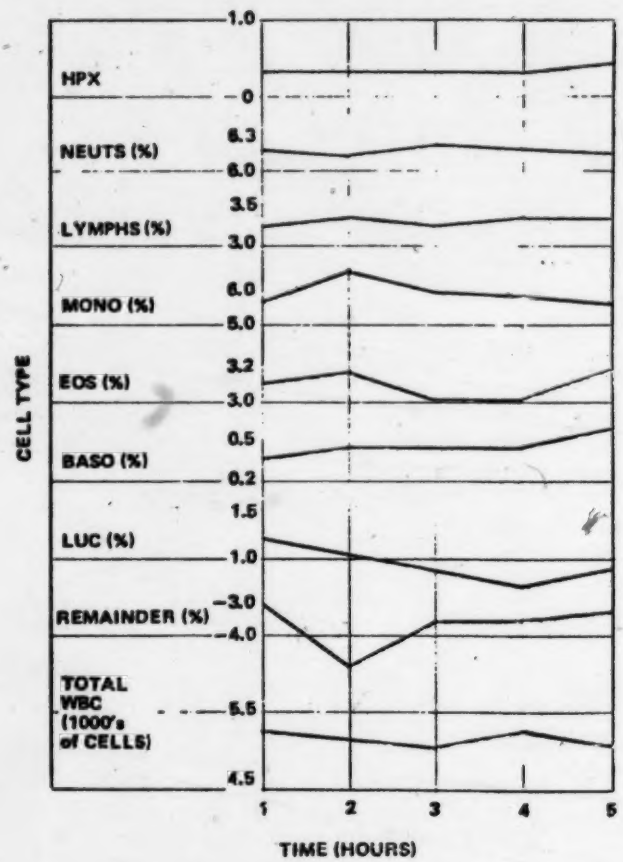


Figure 11-32
DRIFT STUDY

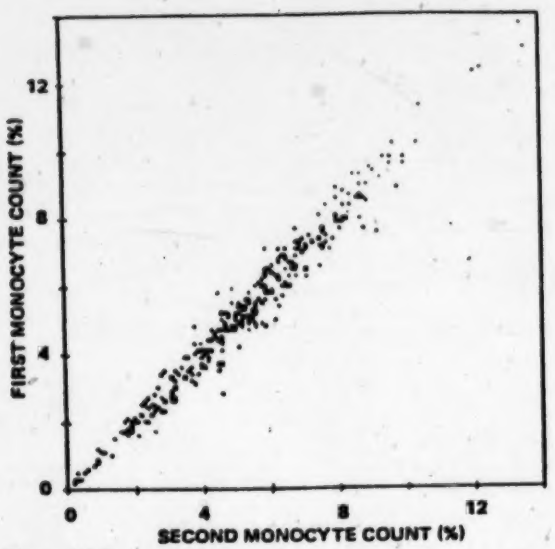


Figure 11-5
CORRELATION OF DUPLICATE MONOCYTE COUNTS – TECHNICON HEMALOG D SYSTEM

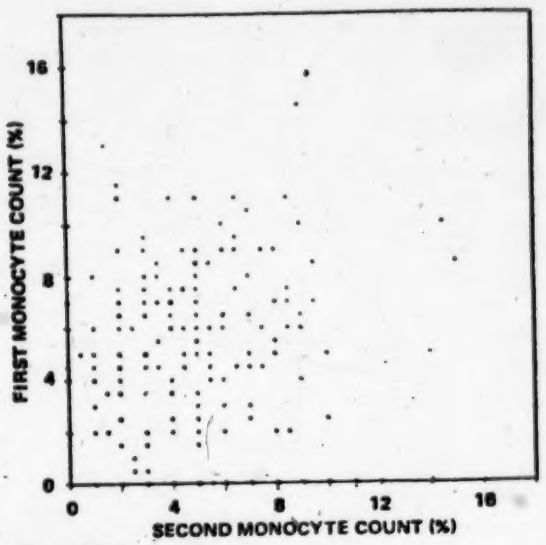


Figure 11-6
CORRELATION OF DUPLICATE MONOCYTE COUNTS – 100-CELL EYE COUNT

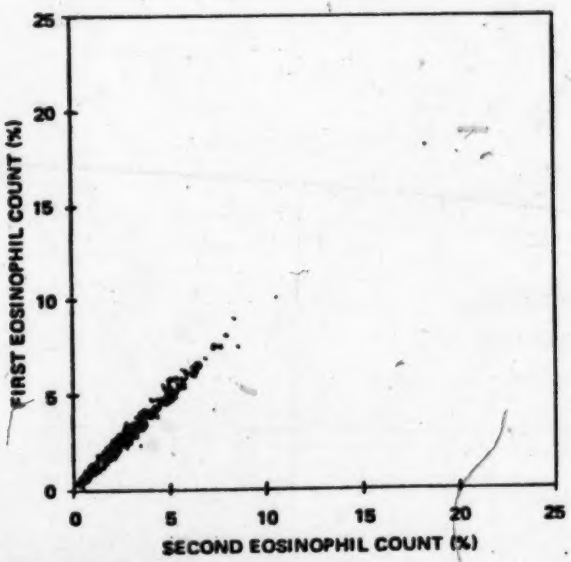


Figure 11-7
CORRELATION OF DUPLICATE EOSINOPHIL COUNTS – TECHNICON HEMALOG D SYSTEM

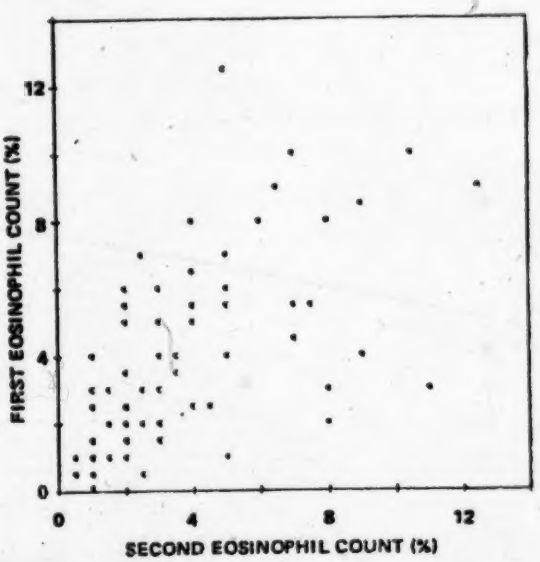


Figure 11-8
CORRELATION OF DUPLICATE EOSINOPHIL COUNTS – 100-CELL EYE COUNT

APPENDIX 'D'

ACTIVITIES QUESTIONAIRE

UNIVERSITY OF ALBERTA

SWIMMING RESEARCH
PROJECT

Activities Questionaire

The following questions relate to your activities during the past summer prior to the commencement of swimming training in October, 1978.

1. Did you take part in regular exercise/activity during the six weeks (or more) before October 2? ... ... YES/NO

2. Would you rate your activity as high, moderate or low?
   i.e. HIGH: 4 sessions per week or more H
   MODERATE: 2-3 sessions per week or more M
   LOW: 1 or less sessions per week L

3. Briefly describe the nature of your activity, e.g. recreational cycling once per week, or hard running 4 times per week, etc.

_____

_____

_____

_____

_____

_____

_____

NAME: _____ (print clearly)

APPENDIX 'E'

GENERAL INFORMATION ON THE TRAINING PROGRAM

EXAMPLE OF A
TRAINING SCHEDULE

DATE: WED. OCT. 18, '78

MORNING SESSION

HWL GROUP

1. 32x50 FLY/FREE
2. 6x400 FREE
3. 400 B/K KICK
4. 400 F/S KICK
5. 4x50 SPRINTS
6.
7.
8.

TOTAL 5000 yards

LWL GROUP

1. 16x50 FREE
2. 8x200 FREE
3. 3x200 KICK
4.
5.
6.
7.
8.

TOTAL 3000 yards

AFTERNOON SESSION

HWL GROUP

1. 16x50 2 of each stroke
2. 4x100 1 of each stroke
3. 200 east FREE
4. 8x200 BREAST
5. 1500 FREE
6. 16x25 FLY/FREE
7.
8.

TOTAL = 4900 yards

LWL GROUP

1. 8x50 2 of each stroke
2. 4x100 1 of each stroke
3. 200 easy FREE
4. 6x200 BREAST
5. 1500 FREE
6.
7.
8.

TOTAL = 3700 yards

DAILY TOTAL: 9900 yards

DAILY TOTAL: 6700 yards

APPENDIX 'F'

THE *SPSS COMPUTER PROGRAMS

```
1     $SIGNON HAQU P=150 T=1M
2     $RUN *SPSS 8=MIK3
3     RUN NAME            ANALYSIS OF BLOOD SAMPLES
4     TASK NAME           BREAKDOWN OF STATS ON EOS & TWBC
5     VARIABLE LIST       SEX, GRP, DAY, MON, ID#, TEST, EOS, TWBC
6     INPUT MEDIUM        DISK
7     INPUT FORMAT        FIXED(F1.0,F1.0,2F2.0,1X,F3.0,F1.0,21X,F4.2,17X,F4.2)
8     N OF CASES          UNKNOWN
9     COMPUTE             DATE=MON*100+DAY
10    IF                  (GRP EQ 1 OR 2) INTENS=2
11    IF                  (GRP EQ 3 OR 4) INTENS=1
12    IF                  (GRP EQ 1 OR 3) LOAD=2
13    IF                  (GRP EQ 2 OR 4) LOAD=1
14    RECODE              TEST (0=2) (1=1)
15    MISSING VALUES      SEX TO TWBC (0)
16    VALUE LABELS        INTENS LOAD(1) LOW(2) HIGH / SEX (1)MALE (2)FEMALE /
17                        TEST (1)PRE (2)POST/
18    *SELECT IF          (DATE EQ 0918)
19    BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
20
21    OPTIONS             2
22    STATISTICS          ALL
23    READ INPUT DATA
24    *SELECT IF          (DATE EQ 0920)
25    BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
26
27    OPTIONS             2
28    STATISTICS          ALL
29    READ INPUT DATA
30    *SELECT IF          (DATE EQ 0922)
31    BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
32
33    OPTIONS             2
34    STATISTICS          ALL
35    READ INPUT DATA
36    *SELECT IF          (DATE EQ 0925)
37    BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
38
39    OPTIONS             2
40    STATISTICS          ALL
41    READ INPUT DATA
42    *SELECT IF          (DATE EQ 0927)
43    BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
44
45    OPTIONS             2
46    STATISTICS          ALL
47    READ INPUT DATA
48    *SELECT IF          (DATE EQ 0929)
49    BREAKDOWN           TABLES=EOS,TWBC BY SEX BY GRP BY TEST
50
51    OPTIONS             2
52    STATISTICS          ALL
53    READ INPUT DATA
54    *SELECT IF          (DATE EQ 1030)
55    BREAKDOWN           TABLES=EOS, TWBC BY SEX BY  GRP BY TEST
```

```
 56
 57      OPTIONS             2
 58      STATISTICS          ALL
 59      READ INPUT DATA
 60      *SELECT IF          (DATE EQ 1101)
 61      BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
 62
 63      OPTIONS             2
 64      STATISTICS          ALL
 65      READ INPUT DATA
 66      *SELECT IF          (DATE EQ 1103)
 67      BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
 68
 69      OPTIONS             2
 70      STATISTICS          ALL
 71      READ INPUT DATA
 72      *SELECT IF          (DATE EQ 1106)
 73      BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
 74
 75      OPTIONS             2
 76      STATISTICS          ALL
 77      READ INPUT DATA
 78      *SELECT IF          (DATE EQ 1108)
 79      BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
 80
 81      OPTIONS             2
 82      STATISTICS          ALL
 83      READ INPUT DATA
 84      *SELECT IF          (DATE EQ 1101)
 85      BREAKDOWN           TABLES=EOS, TWBC BY SEX BY GRP BY TEST
 86
 87      OPTIONS             2
 88      STATISTICS          ALL
 89      READ INPUT DATA
 90      FINISH
 91      $ENDFILE
 92      SIGNOFF
END OF FILE
```

```
  1     $SIGNON HAQU
  2     $RUN *SPSS 8=MIK3
  3     RUN NAME            ANALYSIS OF BLOOD SAMPLES
  4     TASK NAME           ANOVA ON TRAINING EOS & TWBC
  5     VARIABLE LIST       SEX, GRP, DAY, MON, ID#, TEST, EOS, TWBC
  6     INPUT MEDIUM        DISK
  7     INPUT FORMAT        FIXED(F1.0,F1.0,2F2.0,1X,F3.0,F1.0,21X,F4.2,17X,F4.2)
  8     N OF CASES          UNKNOWN
  9     COMPUTE             DATE=MON*100+DAY
 10     IF                  (GRP EQ 1 OR 2) INTENS=2
 11     IF                  (GRP EQ 3 OR 4) INTENS=1
 12     IF                  (GRP EQ 1 OR 3) LOAD=2
 13     IF                  (GRP EQ 2 OR 4) LOAD=1
 14     RECODE              TEST (0=2) (1=1)
 15     MISSING VALUES      SEX TO TWBC (0)
 16     VALUE LABELS        INTENS LOAD(1) LOW(2) HIGH / SEX (1)MALE (2)FEMALE/
 17                         TEST (1)PRE (2)POST/
 18     *SELECT IF          (DATE GT 1002 AND SEX EQ 1)
 19     ANOVA               EOS TWBC BY GRP (1,4) TEST (1,2)
 19.5
 19.7
 19.8
 19.9
 20     STATISTICS          ALL
 21     READ INPUT DATA
 22     SAVE FILE           MIKMAST2
 23     FINISH
 24     $ENDFILE
 25     SIGNOFF
END OF FILE
```

```
  1     $SIGNON HAQU
  2     $RUN *SPSS 8=MIK4
  3     RUN NAME            ANALYSIS OF BLOOD SAMPLES
  4     TASK NAME           ANOVA ON BASAL MVO2 %BF EOS & TWBC
  5     VARIABLE LIST       GRP,MVO1,MVO2,TWBC,EOS,%BF1,%BF2
  6     INPUT MEDIUM        DISK
  7     INPUT FORMAT        FIXED(F1.0,3X,F4.1,1X,F4.1,1X,F4.2,1X,F5.3,1X,
  8                         F4.1,1X,F4.1)
  9     N OF CASES          UNKNOWN
 10     IF                  (GRP EQ 1 OR 2) INTENS=2
 11     IF                  (GRP EQ 3 OR 4) INTENS=1
 12     IF                  (GRP EQ 1 OR 3) LOAD=2
 13     IF                  (GRP EQ 2 OR 4) LOAD=1
 14     VALUE LABELS        INTENS LOAD(1) LOW(2) HIGH /
 15
 16     ONEWAY              MVO2 %BF2 BY GRP(1,4)
 17
 18
 19
 20
 21     STATISTICS          ALL
 22     READ INPUT DATA
 23     SAVE FILE           MIKMAST2
 24     FINISH
 25     $ENDFILE
 26     SIGNOFF
END OF FILE
```

APPENDIX 'G'

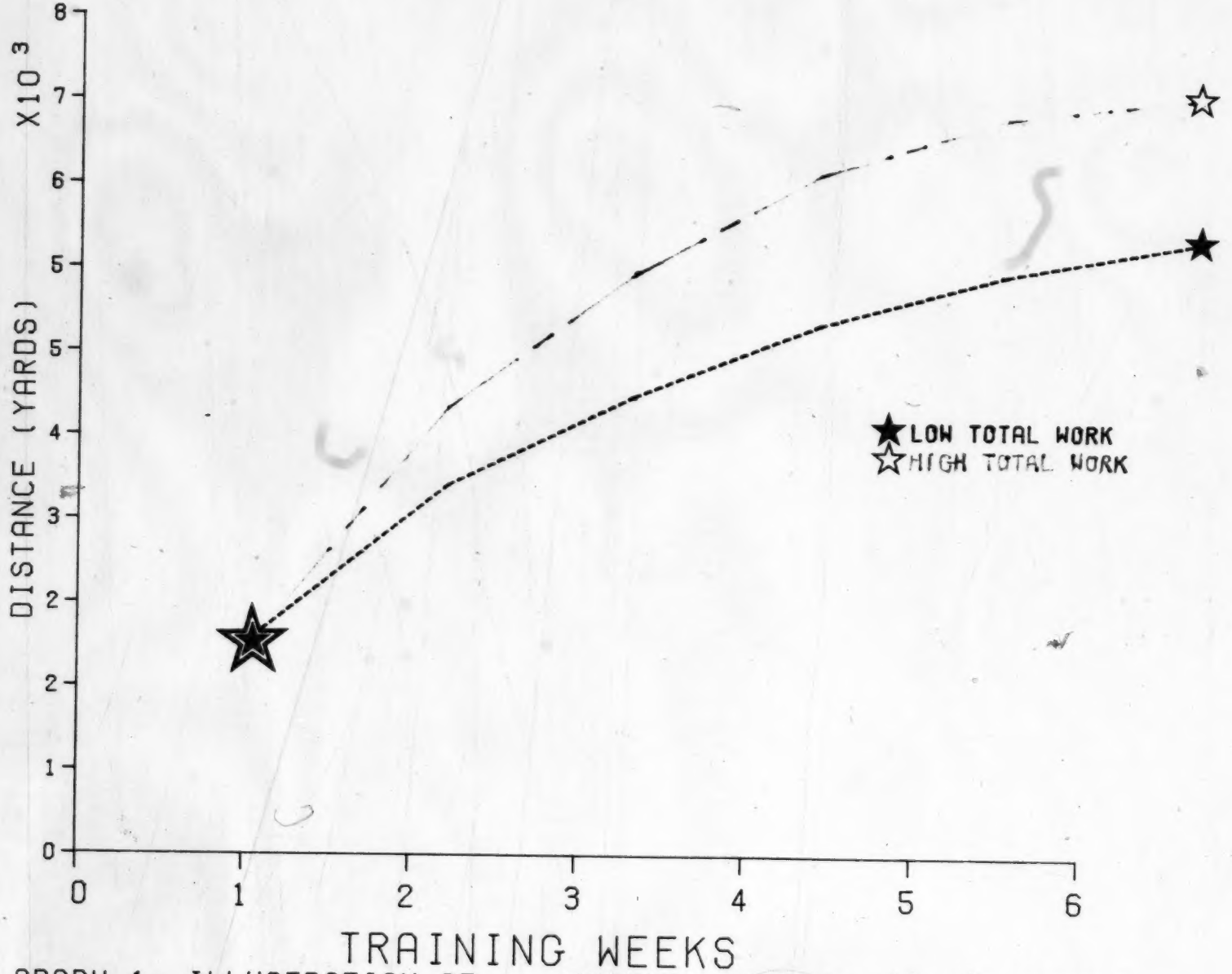
THE *PLOTLIB GRAPH PROGRAMS

PROGRAM FOR PLOTTING GRAPHS

```
      DIMENSION X(20),Y1(20),Y2(20),Y3(20),Y4(20)
      REAL LEG X(4), LEG Y(4)
      DATA X/1.,2.,3.,4.,5.,6.,7.,8.,9.,10.,11.,12.,13.,14.,15.,
     116.,17.,18.,0.C,1.0/
     1,    Y1/2467.,2567.,2237.,2512.,2400.,2150.,2025.,2300.,1900.,
     12171.,1875.,1583.,1443.,1100.,0967.,1229.,1550.,1929.,
     1600.,260./
     1,    Y2/4050.,2683.,2343.,2286.,2657.,2143.,2043.,1750.,1529.,
     12050.,1586.,0983.,1300.,1300.,1071.,1280.,1143.,1633.,600.,
     1260./
     1,    Y3/2600.,2050.,1744.,1700.,2022.,1867.,1589.,1043.,1614.,
     11100.,1433.,1086.,0933.,0800.,0633.,0800.,1060.,1600.,600.,
     1260./
     1,    Y4/4450.,3020.,2740.,3460.,2700.,2040.,0600.,2450.,2900.,
     12400.,1875.,1650.,2700.,1267.,1585.,2800.,2200.,2150.,600.,
     1260./
      CALL PLOTS
      CALL METRIC(1)
      CALL SYMBOL(13.0,7.5,0.4,'GRAPH 2: GROUP MEANS OF',0.0,23)
      CALL SYMBOL(20.0,7.5,0.4,'POST-EX EOSINOPHIL COUNTS',0.0,18)
      CALL NEWPEN (1)
      CALL PLOT(10.,10.,-3)
      CALL AX2EP(1.,3,0,0,1.)
      CALL AXIS2(0.,0.,'SAMPLE DAYS',-11,20.,0.,0.,1.,1.)
      CALL AX2EP(-.3846,3,0,0,1.)
      CALL AXIS2(0.,0.,'EOS X10-1/MM3',13,15.3,90.0,600.,260.,-.3846)
      CALL PLOT(6.0,0.0,3)
      CALL PLOT(6.0,15.0,2)
      CALL CNTRLN(X,Y1,18,1,1,1)

      CALL SYMBOL(15.0,14.5,0.3,'GROUP ONE',0.0,9)
      CALL DASHLN(X,Y2,18,1,1,1)

      CALL SYMBOL(15.0,14.0,0.3,'GROUP TWO',0.0,9)
      CALL NEWPEN (2)
      CALL LINE(X,Y3,18,1,1,139)
      CALL LINE(LEG X(3),LEG Y(3),2,1,1,139)
      CALL SYMBOL(15.0,13.5,0.3,'GROUP THREE',0.0,11)
      CALL NEWPEN (3)
      CALL LINE(X,Y4,18,1,1,1)
      CALL LINE(LEG X(4),LEG Y(2),2,1,1,2)
      CALL SYMBOL(15.0,13.0,0.3,'GROUP FOUR',0.0,10)

      CALL SYMBOL(1.0,1.0,0.3,'BASAL SAMPLES',0.0,13)
      CALL SYMBOL(8.0,1.0,0.3,'TRAINING SAMPLES',0.0,16)
      CALL PLOT(0.,0.,999)
      STOP
      END
END OF FILE
```

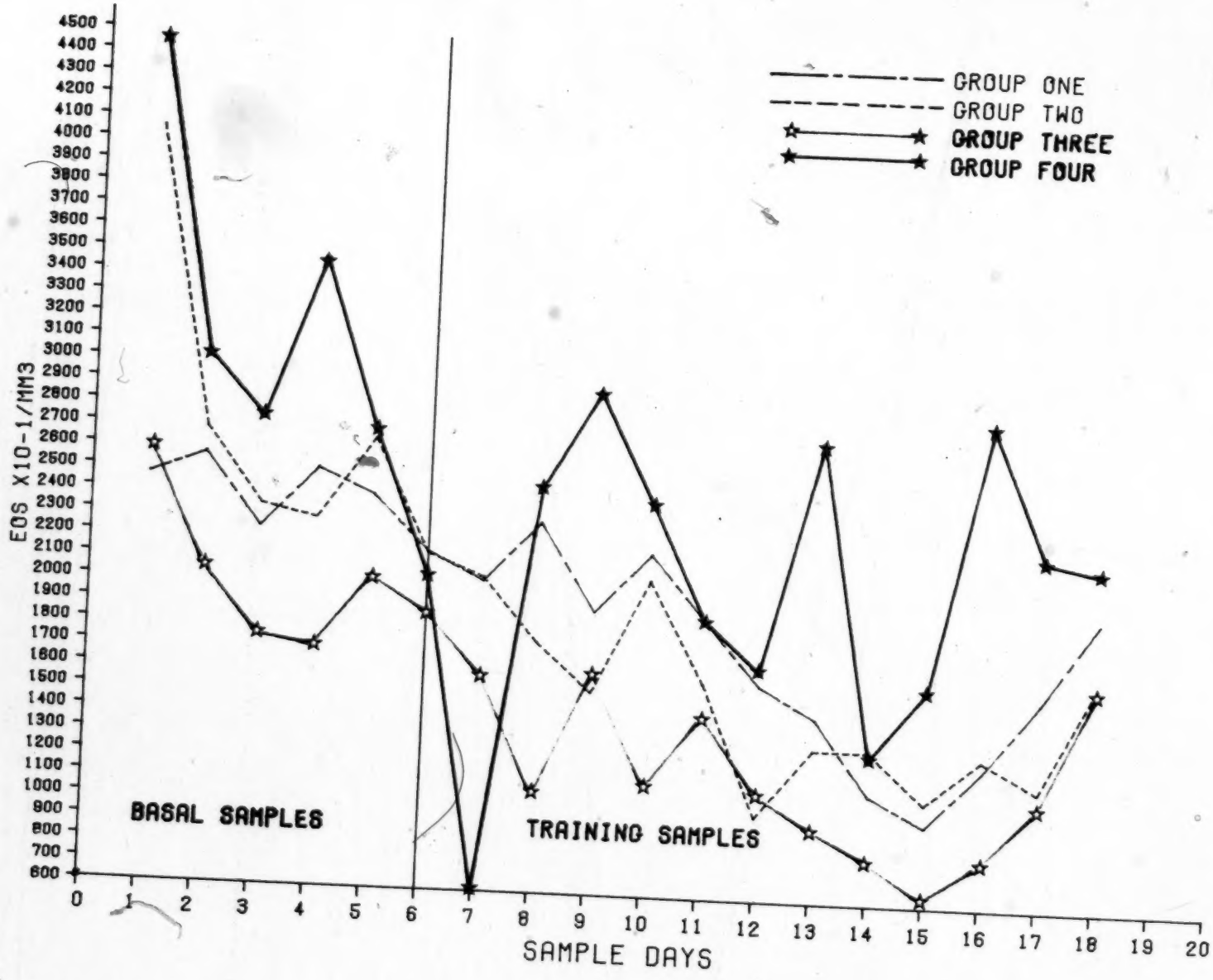
APPENDIX 'H'

GRAPHS



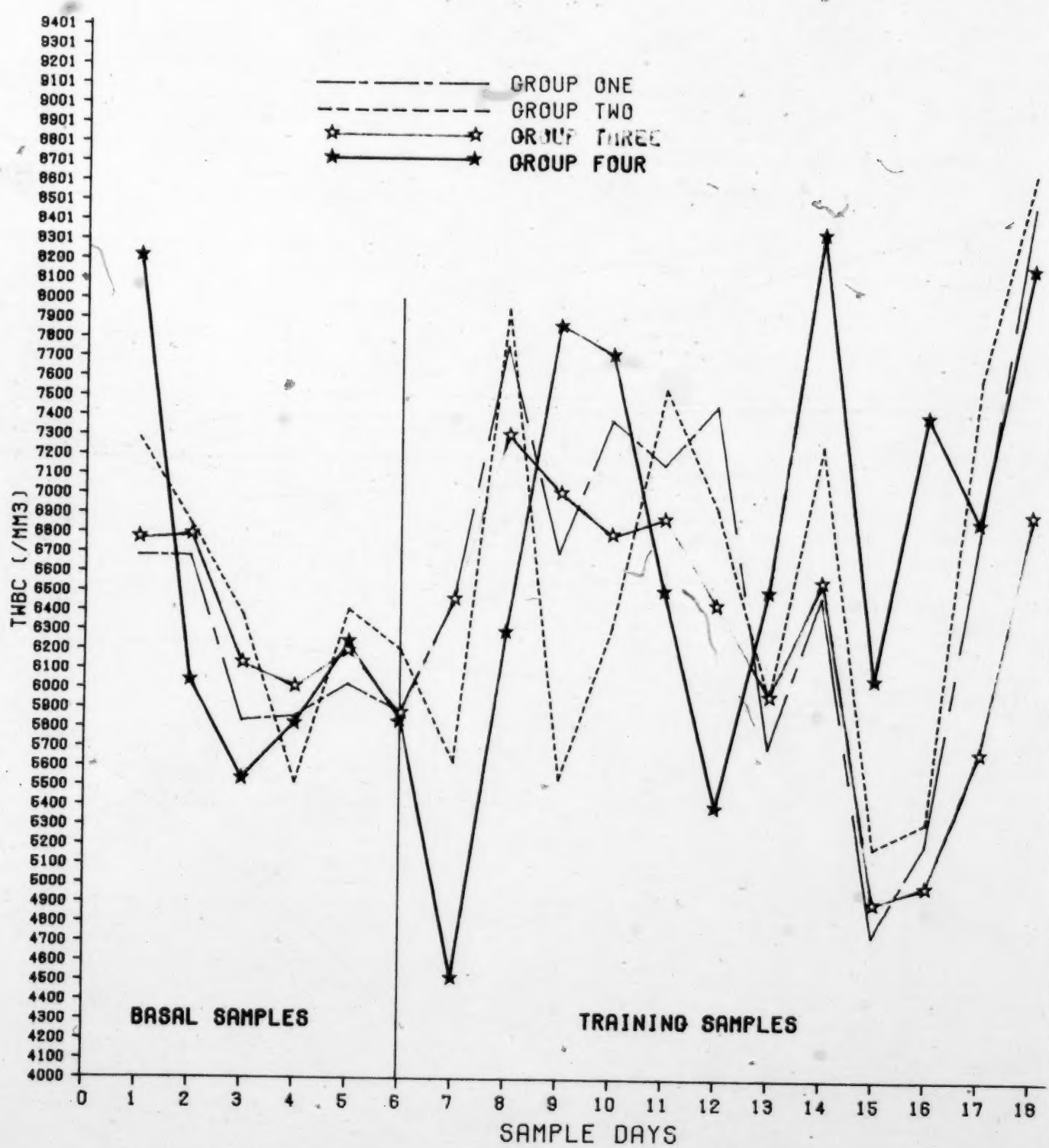
GRAPH 1: ILLUSTRATION OF GRADUAL APPLICATION OF STRESS

Leaf 105 omitted in page numbering



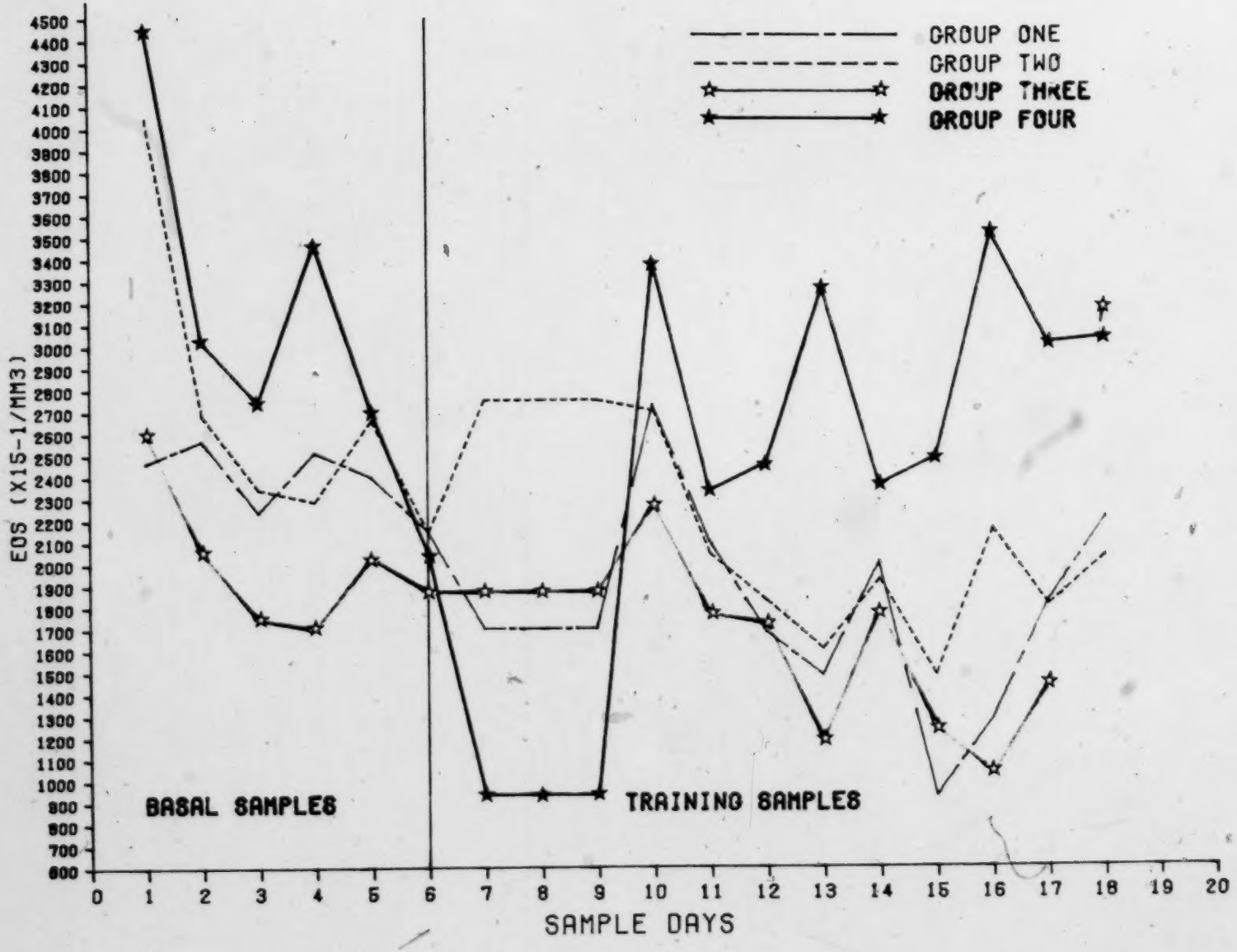
GRAPH 2: GROUP MEANS OF POST-EX EOSINOPHIL COUNT

Leaf 107 omitted in page numbering



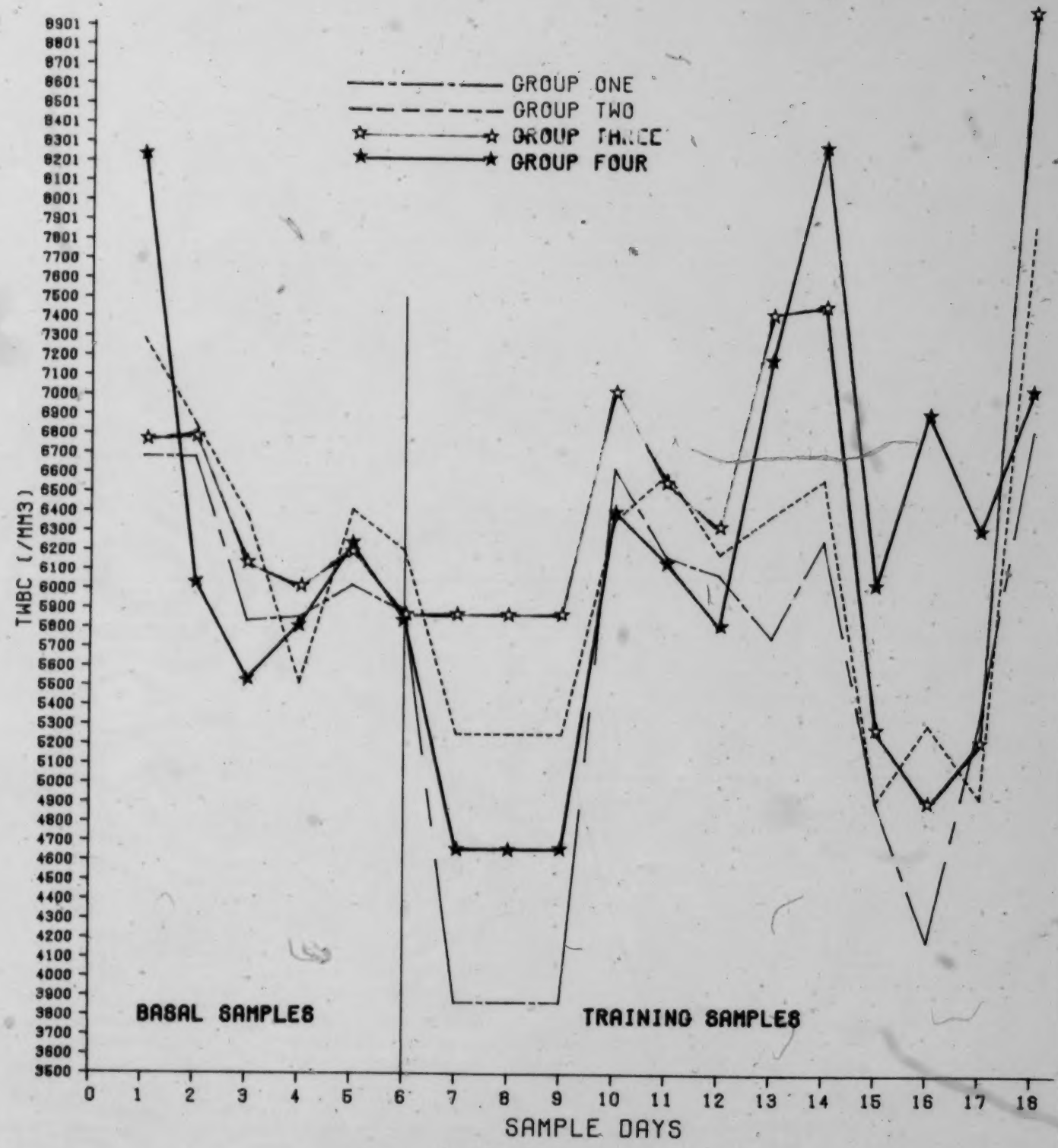
GRAPH 3: POST-EXERCISE TWBC

Leaf 109 omitted in page numbering



GRAPH 4: GROUP MEANS OF PRE-EX EOSINOPHIL COUNTS

Leaf 111 omitted in page numbering



GRAPH 5: PRE-EXERCISE TWBC

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APPENDIX 'I'

FORTRAN PROGRAM FOR DETERMINING TRAINING INTENSITY

COMPUTER PROGRAM TO DETERMINE TRAINING INTENSITY BASED ON PERSONAL BEST TIMES

```
0:            21]
1:           ←]
2: ent "...  UPDA
 TES ? [1 = YES]
 ",r0
3: if r0=1;sto
 29
4: prt "--------
 --------";spc
5: ent "STUDENT
 I.D. TO BE RECO
 VERED",K
6: ent "PERCENT
 CHANGE IN PERFO
 RMANCE",O
7: for A=1 to 40
8: fxd 0;if A[A,
 1]=K;prt "I.D.
 ",A[A,1];fxd 2
9: if A[A,1]=K;
 prt "% CHANGE",
 O;spc ;sto 12
10: next A
11: if A=41;prt
 "I.D. NOT FOUND
 .";K;spc ;sto
 4
12: for B=2 to
 21
13: if A[A,B]=0;
 0→E→F→G→H;sto
 20
14: if A[A,B]>=6
 0;A[A,B]/60→E;
 int(E)→E;A[A,
 B]-60E→F
15: if A[A,B]<60
 ;A[A,B]→F
16: A[A,B]O/100+
 A[A,B]→U
17:          ..'U
 60
 U-60G→H
18: if U<60;U→H
19: fmt 5f2.0
20: wrt 16,3-1,
 "  ",E,":",F,"
  ",G,":",H
21: if B=5;spc
22: if B=9;spc
23: if B=13;spc
24: if B=17;spc
25: if B=19;spc
26: next B
27: spc ;prt "--
 --------------
 ";spc
28: sto 4
29: ent "SUBJECT
  I.D. ?",M
30: for V=1 to
 40
31: if M=A[V,1];
 V→M;jmp 2
32: next V
33: ent "EVENT
 #",N;N+1→N
34: ent "VALUE
 TO BE ENTERED
 =",Z
35: Z→A[M,N]
36: ent "MORE
 UPDATES ? [1 =
 YES]",r1
37: if r1=1;sto
 29
38: if r1#1;rcf
 3,A[*];prt "UPD
 ATED";spc ;sto
 4
*18550
```

EXAMPLES OF OUTPUT FROM TRAINING INTENSITY COMPUTER PROGRAM

I.D.		23
% CHANGE		20.00
1	0:35	0:42
2	1:17	1:33
3	2:50	3:24
4	6:30	7:48
5	0: 0	0: 0
6	1:27	1:44
7	3:12	3:51
8	0: 0	0: 0
9	0: 0	0: 0
10	1:43	2: 3
11	3:34	4:17
12	0: 0	0: 0
13	0: 0	0: 0
14	1:41	2: 1
15	4:11	5: 1
16	0: 0	0: 0
17	3:12	3:50
18	7: 1	8:25
19	0: 0	0: 0
20	0: 0	0: 0

I.D.		38
% CHANGE		12.00
1	0:36	0:40
2	1:16	1:25
3	2:39	2:58
4	5: 8	5:45
5	0: 0	0: 0
6	1:32	1:43
7	2:57	3:19
8	0: 0	0: 0
9	0: 0	0: 0
10	1:19	1:29
11	2:51	3:12
12	0: 0	0: 0
13	0: 0	0: 0
14	1:28	1:33
15	2:48	3: 8
16	0: 0	0: 0
17	2:53	3:14
18	5:46	6:28
19	0: 0	0: 0
20	0: 0	0: 0